

THE EFFECT OF BEHAVIOURAL INTERVENTIONS FOR SLEEP PROBLEMS ON
SECONDARY OUTCOMES IN CHILDREN WITH ASD AND THEIR PARENTS

A thesis in partial fulfilment of the requirement for the Degree of
Master of Science in Child and Family Psychology
At the University of Canterbury

By Jemma Vivian

2018

University of Canterbury, New Zealand

Table of Contents

List of Tables	6
List of Figures	7
Acknowledgements.....	8
Abstract	9
Chapter 1	10
Introduction and Literature review	10
Autism Spectrum Disorder	11
Definition.	11
Prevalence of ASD.....	12
Etiology of ASD	14
Genetic factors.	15
Environmental factors.....	16
Prenatal factors.....	16
Perinatal factors.	17
Epigenetic factors.....	17
Developmental Course of ASD	18
Challenging Behaviours in Children with ASD.....	20
Externalizing behaviours	20
Internalising behaviours.....	21
Sleep in Children with ASD	22
The function of sleep.	22
Types of Sleep Disturbances.....	23
Dyssomnias.....	23
Insomnia related to sleep onset associations.....	23
Insomnia related to excessive time in bed.	24
Limit setting behavioural insomnia.	24
Sleep apnoea/ sleep-disordered breathing.....	25
Parasomnias.	25
Co-sleeping.	26
Prevalence of Sleep Problems in ASD.....	27
Trajectory of Sleep Problems.....	28
Causes of Sleep Problems in Children with ASD.....	28

Environmental factors.....	29
A behavioural model of sleep disturbance.....	29
Familial factors.	31
Psychological/behavioural characteristics.	31
Diagnostic features of ASD.	32
Melatonin levels and circadian rhythms.	34
Co-morbidities/medical issues.	34
Common Sleep Treatments.....	35
Pharmacological treatments.	35
Sleep hygiene.	36
Behaviourally Based Treatments for Sleep Problems in Children with ASD and the Evidence of Effectiveness in both TD Children and Children with ASD.	38
Scheduled awakenings.	39
Faded bedtime.	40
Sleep restriction.	40
Standard extinction.	41
Graduated extinction.	42
Secondary Effects of Sleep Disturbance in Children with ASD.....	44
Child outcomes.	44
Secondary Effects of Sleep Disturbance in parents of Children with ASD.....	60
Parental Outcomes	60
Limitations of Existing Research.....	69
Rationale	71
Chapter 2.....	73
Methodology	73
The Current Study in the Context of the Larger Study	73
The sleep Research Team	73
Ethics and Consent.....	74
Design	74
Data Analysis	75
Participants.....	75
Recruitment.....	76
Inclusion/exclusion criteria.	76
Inclusion/exclusion criteria for the current study.	76

Participants for the current Study.	76
Measures	78
Sleep measures.....	78
Psychometrics.	78
Sleep outcome measures.....	78
Parent-wellbeing measures.	80
Measures of daytime functioning.....	82
Treatment acceptability measures.....	84
Procedure/study phases	85
Clinical interview.....	85
Assessment.....	86
Baseline.....	86
Intervention.....	86
Maintenance.....	87
Follow-up.....	87
Results.....	89
Chapter 3	89
Quality of Data.....	89
Child Sleep and CSHQ, GARS-3, and CBCL Measures	91
Parent PSQI, RQI and DASS-21 Outcome Measures	93
The Relationship Between the CSHQ, CBCL, GARS-3, DASS-21, PSQI and the RQI	99
Modified Brinley Plots Interpretation	103
Modified Brinley Plots – Child Outcomes.....	105
CSHQ.....	106
CSHQ Abbreviated version.	107
CBCL Externalising Behaviours.....	107
CBCL Total Problem Behaviours.....	109
GARS-3.....	111
Modified Brinley Plots – Parent Outcomes	112
RQI.....	112
PSQI.....	113
DASS-21.....	116
Treatment Acceptability- TARF-R	117

Chapter 4.....	119
Discussion	119
Research Questions	119
Study Findings	120
Research Question One.....	120
Research Question Two	124
Strengths of the study.....	130
Limitations	130
Future Directions	132
Conclusion	133
References	135
Appendices.....	182
Appendix A: Child Information Sheet	182
Appendix B: Child Consent Form	184
Appendix C: Parent Information Sheet.....	186
Appendix D: Parent Consent Form.....	189
Appendix E: Sleep Diary Template	191

List of Tables

Table 1. Summary of Participant Characteristics	77
Table 2. Summary of Study Phases	88
Table 3. Means and Standard Deviations for Childrens' Outcome Measures	91
Table 4. Means and Standard Deviations for Parents' Outcome Measures.....	93
Table 5. Pearson Product Moment Correlation of Pre- Intervention Psychometric Scores.....	99
Table 6. Pearson Product Moment Correlation of CSHQ Change Scores and Parent and Child Outcome Measures.....	102
Table 7. Post-intervention Treatment Acceptability Scores from TARF-R	117

List of Figures

<i>Figure 1.</i> Distribution of pre- and post-intervention child outcomes scores on the CSHQ, GARS-3 and the CBCL.	97
<i>Figure 2.</i> Distribution of pre- and post-intervention parent outcome scores on the RQI, PSQI and the Depression, Anxiety and Stress subscales of the DASS-21.....	99
<i>Figure 3:</i> Interpretation of the graph zones in modified Brinley Plots.....	103
<i>Figure 4.</i> Modified Brinley plot showing change from pre- to post- intervention on the full-scale CSHQ.....	105
<i>Figure 5.</i> Modified Brinley plot showing change from pre- to post- intervention on the abbreviated version of the CSHQ.	106
<i>Figure 6.</i> Modified Brinley plot showing change from pre-intervention to post-intervention on Externalising Behaviours on the CBCL outcome measure... ..	107
<i>Figure 7.</i> Modified Brinley plot showing change from pre- to post-intervention on Total Problem Behaviours on the CBCL outcome measure.. ..	109
<i>Figure 8.</i> Modified Brinley plot showing change from pre- to post-intervention on the GARS-3 outcome measure.	110
<i>Figure 9.</i> Modified Brinley plot showing change from pre-intervention to post-intervention on the RQI outcome measure for 18 parents who were in a relationship at the time of assessment.....	112
<i>Figure 10.</i> Modified Brinley plot showing change from pre-intervention to post-intervention on the PSQI outcome measure.	113
<i>Figure 11.</i> Modified Brinley plot showing change from pre- to post-intervention on the subscales of the DASS-21 outcome measure.....	115

Acknowledgements

Firstly, I would like to express my sincere gratitude to my supervisors, Dr Laurie McLay and Associate Professor Karyn France for their support throughout my time with the sleep research team and in the writing of my thesis. Laurie, I would like to thank you for your feedback and valuable time reading my drafts. Karyn, thank you for checking in on me and making sure I was always on track. A big thank you to Professor Neville Blampied also, for his help with analysing the data and the modified Brinley plots. Lastly, I cannot begin to express my gratitude and appreciation of the unwavering support from my family, friends and partner throughout writing my thesis. Thank you for always reminding me to laugh no matter how stressed I was.

Abstract

Sleep problems are highly prevalent in children with Autism Spectrum Disorder (ASD) and are one of the most common sources of concern for parents. A small number of studies have investigated the negative secondary effects associated with sleep problems. However, there has been limited research looking at the outcomes of successful behavioural sleep interventions on children's daytime behaviour and parental well-being. The purpose of this study was to investigate whether improved sleep resulting from behaviourally based sleep interventions had any effect on children's daytime behaviour and parental well-being. Twenty-four children participated, aged between 3 and 14 years including their parents. The design of this study was a single-case multiple baseline across participants with pre- and post-intervention measures. In response to treatment, the study did not find consistent improvements in children's daytime functioning or parental wellbeing across all participants, as measured by the CSHQ, GARS-3, CBCL, DASS-21, PSQI and RQI. However, the majority improved on these measures and some to a clinically significant degree. Behaviourally-based sleep interventions were found to be effective in improving marital satisfaction, parent sleep quality as well as levels of depression, anxiety and stress among parents and decreasing ASD severity and problem behaviours (e.g., externalising behaviour) in children. However, deteriorations were also evident, with some parent's deteriorating to a clinically significant degree on these outcomes. The present findings add to the limited literature investigating pre- and post- measures of secondary outcomes associated with sleep difficulties in the ASD population. Future research may address the use of objective and subjective measures as well as participant specific variables in response to treatment.

Chapter 1

Introduction and Literature review

Sleep problems in children with Autism Spectrum Disorder (ASD) are common and include circadian rhythm disturbances, bedtime resistance, prolonged sleep onset, co-sleeping, night waking, early morning waking and short night-time sleep duration (Vriend, Corkum, Moon, & Smith, 2011). These sleep problems can have profound negative effects on children's daytime functioning, adaptive behaviour, challenging daytime behaviour, repetitive behaviour/stereotypy, physical health, well-being and social development (Adams, Matson, Cervantes, & Goldin, 2014; Delahaye et al., 2014; Hollway, Aman, & Butter, 2013; Hundley, Shui, & Malow, 2016; Mayes & Calhoun, 2009; Mazurek & Sohl, 2016; Taylor, Schreck, & Mulick, 2012; Tudor, Hoffman, & Sweeney, 2012). These problems not only directly affect the children themselves but can also affect family functioning, marital satisfaction, and maternal mental health and well-being (Boergers, Hart, Owens, Streisand, & Spirito, 2007; Chu & Richdale, 2009; Doo & Wing, 2006; Hollway & Aman, 2011; Lopez-Wagner, Hoffman, Sweeney, Hodge, & Gilliam, 2008; Meltzer & Mindell, 2007).

The aim of the current study is to assess whether improvement in the sleep of children with ASD will result in changes in children's daytime behaviour, ASD severity, parental sleep quality and well-being as measured pre- and post-intervention. To date, the majority of research has focussed on associations between sleep disturbance and children's daytime behaviour, ASD symptomatology, learning, anxiety, adaptive functioning, internalising and externalising behaviours in addition to parental stress, depression, anxiety and sleep quality. However, less is known about the effects of resolving sleep problems in children with ASD and how this affects these aspects of children's daytime behaviour as well as parent and child well-being.

In this chapter, ASD and associated challenging behaviours (e.g. externalising and internalising behaviours) are examined. Parental wellbeing associated with ASD are also discussed. Sleep in children with ASD is explored including likely causes and types of sleep problems. Common behaviourally based sleep treatments and the effectiveness of these sleep treatments in children with ASD are introduced. Lastly, the secondary effects of sleep disturbance in children with ASD are discussed. The limitations of existing research studies are explained which demonstrates the significance of continuing research in this area.

The literature review will explore existing research into secondary outcomes of sleep disturbance in children with ASD including externalising, internalising and self-injurious behaviour (SIB), adaptive functioning, ASD symptomatology, anxiety, cognitive functioning, academic achievement, physical health and Health-Related Quality of Life (HRQoL). As well as secondary outcomes of sleep disturbance in parents of children with ASD including stress, anxiety, depression, sleep quality, relationship satisfaction and the impact on the unaffected sibling. This will provide a greater understanding of sleep problems in children with ASD and the impact these sleep problems have.

Autism Spectrum Disorder

Definition. ASD is a developmental disorder characterised by persistent deficits in social communication and repetitive and stereotyped interests and behaviours (American Psychiatric Association, 2013; Cohen, Conduit, Lockley, Rajaratnam, & Cornish, 2014). Impairments in social interaction and communication include deficits in social reciprocity, difficulties with developing and maintaining relationships and non-verbal communicative behaviours used for social interaction. Behavioural symptomatology includes repetitive or stereotyped motor movements (e.g., lining up toys), insistence on sameness, adherence to routines and ritualistic like patterns of nonverbal behaviour (e.g., a need to eat the same food everyday), highly fixated and restricted interests, and unusual interest in sensory aspects of

the environment (e.g., adverse response to specific sounds) (American Psychiatric Association, 2013; Chaudhuri & Chatterjee, 2015). Symptoms must be present in the early developmental period and must cause clinically significant impairment in daily functioning (American Psychiatric Association, 2013).

The features and severity of ASD can vary widely from person to person and symptoms often change as children develop. As a result, ASD is a collective term that encompasses a highly heterogeneous disorder (Robinson et al., 2014). The stage at which functional impairment becomes obvious will vary according to characteristics of the individual, e.g., chronological age, the severity of ASD and the environment, hence the term, spectrum. However, levels of support and compensation may mask the severity of the disorder (American Psychiatric Association, 2013). For a diagnosis to occur, the symptoms present must not be better explained by intellectual disability (American Psychiatric Association, 2013). The term ASD now encompasses diagnoses referred to in the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) as Asperger syndrome and Pervasive Developmental Disorders- Not Otherwise Specified (PDD-NOS) (American Psychiatric Association, 2013).

Prevalence of ASD

According to the Centers for Disease Control and Prevention (American Psychiatric Association, 2013; Christensen et al., 2016), approximately one in 68 children has been identified as having an ASD. Whilst no prevalence data is available in New Zealand, it is estimated that one in every 88 children in New Zealand has ASD (Autism NZ, 2014). Research suggests that the prevalence of ASD has been steadily increasing over time (Idring et al., 2012; Keyes et al., 2012; Lai, Lombardo, & Baron-Cohen, 2014; Matson & Kozlowski, 2011; Srivastava, 2013). The reasons for this are unclear but may include, improved awareness and recognition of ASD, a rise in ASD risk factors (e.g., children born extremely

premature, exposure to heavy metals or other toxins in the environment such as air pollution), younger age of diagnosis and changes in diagnostic concepts and criteria (Charman, 2002; Elsabbagh et al., 2012; Keyes et al., 2012; Lai et al., 2014; Talbott et al., 2015). It is generally accepted that diagnostic criteria for ASD has been broadened and far exceeds the original criteria reported by Leo Kanner (Kanner, 1943; Lyons & Fitzgerald, 2007). Following the initial classification system of ASD, ASD criteria were modified and expanded throughout the subsequent versions of the DSM up until its current issue (DSM-V) (American Psychiatric Association, 2013). These revisions of the DSM resulted in studies using different criteria when assessing the prevalence of ASD, thus increasing the possibility of a misinterpreted increase (Matson & Kozlowski, 2011).

It is widely reported that rates of ASD are much higher in males than in females. A gender difference in the prevalence of ASD has been documented in studies since the 1960's (Lotter, 1966) and large-scale population-based studies have shown that ASD affects two to three times more males than females (Idring et al., 2012; Kim et al., 2011; Saemundsen, Magnússon, Georgsdóttir, Egilsson, & Rafnsson, 2013). A recent meta-analysis conducted by Loomes, Hull, and Mandy (2017) corroborates this data, indicating that among diagnosed cases, there are four boys for every girl on the autism spectrum (American Psychiatric Association, 2013; Loomes et al., 2017). There are several possible explanations for this variation across genders. Firstly, males and females may have different phenotypic symptom presentations. For example, males with ASD show more externalising behaviours such as aggression, hyperactivity and reduced prosocial behaviour (Altman & Turk, 2016; Mandy et al., 2012; Werling & Geschwind, 2013). Typical externalising behaviours in males may be more disruptive in the home and school environment and trigger a clinical evaluation and diagnosis compared to females. By contrast, females are reported to show greater internalising symptoms such as depression, anxiety and other emotional challenges, which

may go undetected during assessment (Altman & Turk, 2016; Frazier, Georgiades, Bishop, & Hardan, 2014; McLennan, Lord, & Schopler, 1993; Werling & Geschwind, 2013; Wijngaarden-Cremers et al., 2014; Zwaigenbaum et al., 2012). Another possible explanation for these gender differences is that the diagnostic criteria are based on symptoms seen in males while females require more severe and complex autistic, behavioural and cognitive symptoms to be diagnosed with ASD (Bargiela, Steward, & Mandy, 2016; Frazier et al., 2014). Females usually show reduced social impairments as compared to males, thus it may be that higher levels of social ability in females prevent a full diagnosis of ASD, especially for those that are high functioning (Halladay et al., 2015). Furthermore, females diagnosed with ASD are more likely to show accompanying intellectual disability, suggesting that females who do not exhibit language or intellectual impairment delays may go unrecognised or receive their diagnoses later, indicating a diagnostic bias towards males (American Psychiatric Association, 2013; Begeer et al., 2013; Dworzynski, Ronald, Bolton, & Happé, 2012; Giarelli et al., 2010; Lai, Lombardo, Auyeung, Chakrabarti, & Baron-Cohen, 2015). If females are receiving their diagnoses later than boys, the male to female ratio may be higher in younger samples in the literature (Loomes et al., 2017). Finally, females are less vulnerable to developing ASD due to innately protective mechanisms (Lai et al., 2011; Lai et al., 2014; Volkmar, Szatmari, & Sparrow, 1993). Females may develop strategies to cope and adapt to certain situations masking their symptoms and they may present with a modified phenotype which may not be adequately captured by current diagnostic instruments leading to underdiagnosis (Attwood, 2007; Haney, 2016; Kirkovski, Enticott, & Fitzgerald, 2013).

Etiology of ASD

The root cause of ASD remains unknown and is a focus of many research studies. However, we still do not have a consistent understanding of cause, possibly due to the heterogeneity and complexity of the disorder (Fakhoury, 2015). Furthermore, most

researchers today believe that the interplay between genetics and environment is likely to play a role in the etiology of ASD (Bierut et al., 2010; Fakhoury, 2015; Folstein & Rosen-Sheidley, 2001; Hunter, 2005; Karimi, Kamali, Mousavi, & Karahmadi, 2017; Liu, Zhang, Rodzinka-pasko, & Li, 2016; Rutter, 2000).

Genetic factors. Evidence for genetic explanations of ASD is still emerging through an increasing amount of children with ASD being found with high-risk genes and genetic abnormalities (Ronald, Plomin, & Happé, 2006). Research estimates that the heritability of ASD ranges from 37% to 90% based on early twin and family studies. (American Psychiatric Association, 2013; Bailey et al., 1995; Connolly & Hakonarson, 2014; Egger et al., 2014; Ronald & Hoekstra, 2011). Currently, 15% of ASD cases appear to be associated with a known genetic mutation with different variants associated with ASD in different families (American Psychiatric Association, 2013). The remaining cases seem to be polygenic, with hundreds of genetic loci making contributions toward ASD (American Psychiatric Association, 2013). Studies have identified mutations in certain brain-expressed genes that are associated with ASD (Fakhoury, 2015). These genes include serotonergic genes, GABA receptor subunit genes, glutamatergic genes, dopaminergic genes as well as mutations in neuroligins (Fakhoury, 2015; Jamain et al., 2002; Nguyen et al., 2014; Paval, 2017). Recent studies have illustrated the importance of mirror neurons in the neuropathophysiology of ASD (Enticott et al., 2012; Fakhoury, 2015; Oberman et al., 2005). Mirror neurons are brain cells that become active when an individual performs an action and enables individuals to understand the actions of others as well as aids social cognitive functions such as empathy (Enticott et al., 2012; Fakhoury, 2015; Gallese, 2007). These studies have found that a dysfunction of the mirror neuron system may contribute to social and cognitive impairments associated with ASD (Cattaneo, Rizzolatti, & Fabbri-Destro, 2009; Gallese, 2007; Rizzolatti

& Fabbri-Destro, 2010; Williams, Whiten, Suddendorf, & Perrett, 2001). These findings collectively provide evidence of a genetic component to ASD (Fakhoury, 2015).

Environmental factors. In addition to the role of genes, there is increasing evidence that indicates that environmental factors may contribute toward ASD (Fakhoury, 2015; Larsson, Weiss, Janson, Sundell, & Bornehag, 2009; Lyall, Schmidt, & Hertz-Picciotto, 2014). Exposure to environmental pathogens may impact on brain development at different stages including synaptogenesis, myelination, cell differentiation and migration.

Prenatal factors. Aspects of the prenatal environment have been known to increase the risk of ASD, including alcohol and drug use, maternal deficiencies in essential vitamins (e.g., iodine, folic acid, fatty acids), gestational diabetes, gestational bleeding, maternal obesity, advanced maternal and paternal age at birth and certain medications such as serotonin reuptake inhibitor antidepressants (Croen, Grether, Yoshida, Odouli, & Hendrick, 2011; Fakhoury, 2015; Gardener, Spiegelman, & Buka, 2009; Li et al., 2016; Lyall et al., 2014; Tordjman et al., 2014; Xu, Jing, Bowers, Liu, & Bao, 2014).

Prenatal maternal infection and subsequent Maternal Immune Activation (MIA) is also thought to increase the risk for a number of neurodevelopmental disorders, including ASD (Atladóttir, Henriksen, Schendel, & Parner, 2012; Atladóttir et al., 2010; Brown, 2012; Careaga, Murai, & Bauman, 2017; Garay, Hsiao, Patterson, & McAllister, 2013; Jiang et al., 2016; Lee et al., 2015; Lombardo et al., 2017; Patterson, Xu, Smith, & Devarman, 2008; Smolders et al., 2015; Zerbo et al., 2013; Zerbo et al., 2015). MIA refers to the mother's immune system defence response to invading pathogens (Estes, 2016; Parker-Athill & Tan, 2011). During an infection, cytokines are released which tell immune cells where to go and what pathogen to destroy (Deverman & Patterson, 2009). During pregnancy increased levels of inflammatory cytokines resulting from infection are likely mediators of increased risk and

disease development and can affect the foetus by altering the architecture and function of the developing brain (Deverman & Patterson, 2009; Howerton & Bale, 2012; Smolders et al., 2015). There is evidence for this from post-mortem brain samples taken from individuals with ASD that show altered cytokines (Atladóttir et al., 2012; Estes & McAllister, 2015; Vargas, Nascimbene, Krishnan, Zimmerman, & Pardo, 2005; Zerbo et al., 2013).

Additionally, a threefold increased risk of ASD was related to maternal hospitalisations for viral infections during the first trimester of pregnancy and hospitalisation due to bacterial infections during the third-trimester (Atladóttir et al., 2010; Brown, 2012). Behavioural deficits relevant to ASD are a known consequence of MIA (Lombardo et al., 2017; Malkova, Collin, Hsiao, Moore, & Patterson, 2012; Shi, Fatemi, Sidwell, & Patterson, 2003; Smith, Li, Garbett, Mirnics, & Patterson, 2007).

Perinatal factors. Perinatal factors that are thought to contribute toward ASD include low birth weight, prematurity, gestation duration, hypoxia during childbirth, planned caesarean section, abnormal presentation during birth (e.g., breech presentation) and foetal exposure to valproate (American Psychiatric Association, 2013; Guinchat et al., 2012; Klevzon, Gross, & Reichenberg, 2007; Sullivan, 2008; Xu et al., 2014). Additional factors may also include exposure to air pollutants such as automotive exhaust and agricultural pesticides (American Psychiatric Association, 2013; Fakhoury, 2015; Klevzon et al., 2007; Larsson et al., 2009; Sullivan, 2008; Volk, Hertz-Picciotto, Delwiche, Lurmann, & McConnell, 2011).

Epigenetic factors. Several lines of evidence have pointed toward epigenetic factors as contributing to ASD together with genetic variants (Persico & Bourgeron, 2006). Many disorders are a result of a combination of complex interactions between the individual's genetic profile and the environment that they are exposed to (Fakhoury, 2015; Hunter, 2005). Environmental factors can interact with susceptibility genes leading to epigenetic gene

expression changes that may lead to an increased risk of ASD (Fakhoury, 2015; Lyall et al., 2014; Schaevitz & Berger-Sweeney, 2012; Volk et al., 2014). Epigenetic gene modifications can include DNA methylation (epimutation) and numerous changes that impact histone proteins such as acetylation (Fakhoury, 2015; Tordjman et al., 2014; Zhu et al., 2014). Early modifications in DNA methylation could stop the normal development of functional neuron networks (Schaevitz & Berger-Sweeney, 2012). Individuals with ASD regularly have altered levels of DNA methylation in genes that play critical roles during processes of brain development such as synaptogenesis (Nardone et al., 2014). For example, children who had both the MET receptor tyrosine kinase (MET) gene CC genotype and high air pollutant exposure, were at increased risk for ASD compared with individuals who had both CG/GG genotypes and lower air pollutant exposure. This suggests a gene-environment interaction for ASD based on MET genotype and air pollutant exposure (Volk et al., 2014). Also, interactive effects have been found between prenatal maternal infection and certain genetic variants on ASD symptomatology (Mazina et al., 2015). Individuals with ASD associated Copy Number Variants (CNV's) were more likely to be vulnerable to maternal infection or fever during pregnancy. Children that were exposed to maternal infection during pregnancy showed increased rates of restricted and repetitive behaviours as well as social communicative deficits compared to other children with ASD (Fakhoury, 2015; Mazina et al., 2015). ASD is a complex and heterogeneous disorder no single genetic or environmental factor has been identified so far (Fakhoury, 2015). Many environmental, genetic and epigenetic factors are still being explored that contribute to the risk and etiology of ASD (Tordjman et al., 2014; Williams et al., 2014).

Developmental Course of ASD

ASD symptoms are typically recognised in the second year of life (12-24 months of age) though diagnosis is made on average, at around four and a half years of age (Howlin &

Moore, 1997; Zwaigenbaum, 2001). It is currently uncommon for ASD to be diagnosed in children younger than three years of age, yet one population study has noted that early signs can be detected at 18 months (Baron-Cohen et al., 1997). ASD can usually be distinguished from other atypical patterns of development by poor joint attention, lack of eye contact, lack of age-appropriate play and diminished social responsiveness (Zwaigenbaum, 2001). Early diagnosis of ASD is essential for timely therapeutic intervention known to improve developmental outcomes for these children (Zwaigenbaum, 2001).

Two possible developmental courses are predominant in the literature relating to ASD in childhood (Ozonoff, Heung, Byrd, Hansen, & Hertz-Picciotto, 2008; Werner, Dawson, Munson, & Osterling, 2005). The first course of development is an emerging gradual course whereby typical development appears during the first year of life. After this time, parents report concerns which usually involve lack of social interest, delayed language development and unusual social interactions (American Psychiatric Association, 2013; Lord, 1995; Zwaigenbaum et al., 2009). During the second year, unusual repetitive patterns of behaviour often start to appear and abnormal play can become evident (e.g. poor imaginative ability, lining up toys, lack of social engagement, deficit in symbolic play and a tendency toward stereotyped, repetitive non-functional object manipulation) (American Psychiatric Association, 2013; Bauman & Kemper, 2005; Jarrold, Boucher, & Smith, 1993; Rapin & Katzman, 1998; Stanley & Konstantareas, 2007). For these children, symptoms are most often marked in early childhood with developmental gains in some areas later in life (e.g., interests in social interaction) (American Psychiatric Association, 2013).

The second course of development is referred to as a regressive form of ASD, where children lose developmental skills following normal or abnormal development in the first two or three years of life (Werner et al., 2005). In the second year of life, they may lose skills that they have acquired and begin to present with autistic symptomatology (Ozonoff et al., 2008).

The developmental areas that are most obviously affected by regression are social and communication abilities (Ozonoff et al., 2008). Following regression, few children with ASD regain normal development (Cohen-Ophir, Castel-Deutsh, & Tirosh, 2012; Rapin & Katzman, 1998). Whilst these two distinct forms of ASD have been documented, it is important to note that these two patterns do not capture all of the different ways ASD can emerge. For example, some children show mixed features with both early delays and later losses evident (Ozonoff et al., 2008).

Challenging Behaviours in Children with ASD

Children with ASD exhibit behaviours that their family, caregivers or teachers often find challenging. Challenging behaviour is most frequently defined as behaviours that are not culturally or socially acceptable and that are of such intensity and duration that the safety of the individual and others in their environment may be at risk. As a result, this can limit the child's involvement in educational and community activities (American Psychiatric Association, 2013; Emerson, 2001; Sigafos, O'Reilly, & Arthur-Kelly, 2003). ASD is often accompanied by at least one challenging behaviour (Matson & Nebel-Schwalm, 2007).

Externalizing behaviours. Approximately 8% to 68% of children with ASD experience some form of externalising behaviour problems (Farmer et al., 2015; Gadow, DeVincent, Pomeroy, & Azizian, 2004; Georgiades et al., 2011; Hartley, Sikora, & McCoy, 2008; Hill et al., 2014; Lecavalier, 2006; Mattila et al., 2010; Murphy, Healy, & Leader, 2009). The most prevalent forms of challenging behaviour in children with ASD are tantrums, property destruction, aggression, self-injury, sleep difficulties, and stereotypies (Cohen, Yoo, Goodwin, & Moskowitz, 2011; Cohen, Conduit, et al., 2014; Emerson et al., 2001; Green, Gilchrist, Burton, & Cox, 2000; Horner, Carr, Strain, Todd, & Reed, 2002; Jang, Dixon, Tarbox, & Granpeesheh, 2011; Matson, Wilkins, & Macken, 2009; McClintock, Hall, & Oliver, 2003). Challenging behaviours in children with ASD vary with age, gender,

ASD diagnosis and level of intellectual functioning (Benson & Brooks, 2008; Cohen et al., 2011; Hill et al., 2014).

Children with ASD are at increased risk for developing challenging behaviours in part because they can lack functional communicative behaviours and thus, often engage in challenging behaviours to communicate their wants and needs (Conroy, Dunlap, Clarke, & Alter, 2005; Martinez, Werch, & Conroy, 2016). If challenging behaviour is not addressed as these children grow up it may increase in both rate and severity and therefore progress to severe conduct disorders later in life (Conroy et al., 2005).

Internalising behaviours. There are inconsistencies in the literature on the prevalence of internalising disorders in children with ASD. Estimates suggest that the prevalence ranges between 11% to 86% in children with ASD (Ando & Yoshimura, 1979; Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; Leyfer et al., 2006; Muris, Steerneman, Merckelbach, Holdrinet, & Meesters, 1998; Ooi, Tan, Lim, Goh, & Sung, 2011; Strang et al., 2012; White, Oswald, Ollendick, & Scahill, 2009). These behaviours include anxiety, compulsions, obsessions, depressed mood, fears, mood disorders and emotional reactivity (Gadow et al., 2004; Gadow, Devincent, Pomeroy, & Azizian, 2005; Gillott, Furniss, & Walter, 2001; Green et al., 2000; Hartley et al., 2008; Russell & Sofronoff, 2005; Weisbrot, Gadow, DeVincent, & Pomeroy, 2005). Among the most common internalising behaviours are anxiety and depression (Bauminger, Solomon, & Rogers, 2010; Simonoff et al., 2008).

Rates of anxiety and depression symptoms have been reported to be higher in children with ASD in comparison with Typically Developing (TD) children (Bellini, 2004; Gadow et al., 2005; Gadow, Guttman-Steinmetz, Rieffe, & DeVincent, 2012; Hurtig et al., 2009; Kim et al., 2000; Mayes, Gorman, Hillwig-Garcia, & Syed, 2013; Russell & Sofronoff, 2005; Solomon, Miller, Taylor, Hinshaw, & Carter, 2012). Furthermore, rates of suicide ideation

and suicide attempts are higher in comparison with children with intellectual disability, Attention-Deficit/ Hyperactivity Disorder (ADHD), chronic multiple tic disorder and TD children (Brereton, Tonge, & Einfeld, 2006; Gadow et al., 2012; Gillott et al., 2001; Mayes et al., 2013). Increased rates of internalising behaviours and mental health issues in children with ASD are thought to result from traumatic family events, theory of mind, one's awareness of their disability and a complex interaction between cognitive, perceptual, biological environmental and social influences (Kim et al., 2000). For example, perceptual and cognitive disturbances caused by ASD may lead to increased mental health issues because of the stress experienced by these children with their interactions with others around them and the possible negative responses from others (Bellini, 2004; Brereton et al., 2006; Gadow et al., 2005; Gadow et al., 2012; Gillott et al., 2001; Hurtig et al., 2009; Kim et al., 2000; Mayes et al., 2013; Russell & Sofronoff, 2005; Solomon et al., 2012).

Sleep in Children with ASD

The function of sleep. Children spend more time sleeping than engaging in any other activity by the time they go to school, including interacting with others, exploring and playing (Wiggs, 2007). Sleep is a dynamic and cyclic brain activity that affects every aspect of a child's development. Therefore, good quality sleep is critical for healthy development, well-being and optimum daytime functioning (Honomichl, Goodlin-Jones, Burnham, Gaylor, & Anders, 2002). The precise reason why people sleep remains unknown, however, sleep is likely to serve multiple purposes and is essential for aspects of development such as brain maturation, physical growth, learning and health (Wiggs, 2007). In addition, many theories on the purpose of sleep have emphasised psychological and physical restoration, energy conservation, consolidation of memories, brain growth and maintenance of the immune system as important functions of sleep in children (Rechtschaffen, 1998; Wiggs, 2007).

Types of Sleep Disturbances

Sleep disorders have been classified into three major categories, (1) dyssomnias, (2) parasomnias and (3) sleep disorders associated with mental, neurologic or other medical disorders (American Academy of Sleep Medicine, 2014; American Psychiatric Association, 2013; Krakowiak, Goodlin-Jones, Hertz-Picciotto, Croen, & Hansen, 2008).

Dyssomnias. Dyssomnias are the most common category of sleep problems in children with ASD. Dyssomnias are sleep disorders characterised by either the inability to sleep (insomnia) or excessive sleepiness (hypersomnia) (American Academy of Sleep Medicine, 2005). The major groups of dyssomnias include intrinsic (problems arising from within the body), extrinsic (arising from pathological conditions or environment) and circadian rhythm disorders (Thorpy, 2012). Behavioural insomnias are common in the paediatric population and are the most frequently reported sleep problem in children with ASD (Owens, 2017; Richdale, 2013). Behaviourally based insomnia in children with ASD typically presents as irregularity in sleep-wake patterns, night and early morning waking, behavioural problems at bedtime, including unusual bedtime routines and settling difficulties, prolonged sleep onset, bedtime resistance, prolonged night awakenings and higher rates of problematic co-sleeping (Cotton & Richdale, 2006; Malow et al., 2006; Nunes & Bruni, 2015; Richdale, 2001; Spruyt & Curfs, 2015). The most commonly reported sleep problems are with sleep onset and maintenance, including delayed sleep onset latencies, night waking, short night sleep duration and early morning waking (Krakowiak et al., 2008).

Insomnia related to sleep onset associations. Sleep associations are learned behaviours that occur when a child associates a certain environment or behaviour with falling asleep. These can include rocking, parental presence or feeding with a bottle (Mindell, Kuhn, Lewin, Meltzer, & Sadeh, 2006). Bedtime is usually not a problem if a parent can provide these specific sleep cues, however, during the night when the child awakens the child may

not be able to get back to sleep (self-soothe) without the same conditions being available (Meltzer, 2010; Mindell et al., 2006). The awakenings may then be accompanied by crying, the child coming into the parents' room, or screaming until the necessary conditions are provided for the child to fall back asleep. For this reason, it is important that the conditions during sleep onset are readily available to the child during the night without involving parental intervention (Meltzer, 2010; Mindell et al., 2006).

Insomnia related to excessive time in bed. “Too much time in bed” does not fall under the typical classification of insomnia but can be helpful in treating and understanding children's sleep problems (Owens & Moore, 2017). When a child is in bed longer than he or she is able to sleep, sleep problems can start occurring which may involve early morning awakenings or problems with sleep onset (Owens & Moore, 2017). For example, a child whose sleep requirement of 10 hours is exceeded by being in bed for 12 hours per night may cause problems. Adjusting the child's time in bed by referring to age-appropriate ranges for sleep may help (Owens & Moore, 2017).

Limit setting behavioural insomnia. Limit setting is often encountered at bedtime when children may challenge their parents and refuse to go to sleep (Meltzer, 2010; Owens & Moore, 2017; Vriend & Corkum, 2011). Limit-setting insomnia is characterised by certain calls or requests which may involve verbal protests, active resistance, demands of wanting a drink or another story after lights are out in the bedroom (Vriend & Corkum, 2011). This insomnia usually develops from the parent or caregiver's inability or unwillingness to enforce strict bedtime routines and sleep hygiene practices (Glaze, 2004; Meltzer, 2010). This can lead to a decreased total sleep time as a result of prolonged sleep onset (Owens & Moore, 2017). When limits are not enforced by caregivers the problem can often be exacerbated by the child's behaviour, especially children with ASD who seem to have challenging bedtime behaviours and tend to be more noncompliant (Richdale, 2013). A predictable and consistent

routine such as a regular bed-time and relaxing pre-bedtime activities may decrease these behaviours (Owens & Moore, 2017).

Sleep apnoea/ sleep-disordered breathing. Sleep-disordered breathing problems can include Obstructive Sleep Apnoea (OSA), Central Sleep Apnoea (CSA) and hypopnea (Murata et al., 2017). Snoring and sleep apnoea are dyssomnias related to breathing problems during sleep. Those affected by sleep apnoea stop breathing during the night, which disrupts sleep. This thesis focuses on behavioural sleep problems. However, sleep apnoea may be an important exacerbating factor for sleep problems in both typically developing (TD) children and children with ASD; however, it is not the focus of this thesis.

Parasomnias. Parasomnias are undesirable phenomena that occur predominantly during sleep. These include sleep movements, behaviours, emotions, perceptions and autonomic nervous system functioning associated with sleep (Thorpy, 2012). Examples of parasomnias include night terrors, repetitive rhythmic behaviours, sleepwalking, sleep talking and nightmares (Markov, Jaffe, & Doghramji, 2006; Thorpy, 2012). It is not uncommon for more than one parasomnia to be present and they often occur alongside other sleep disorders such as obstructive sleep apnoea (Thorpy, 2012). Parasomnias are divided into four groups: arousal disorders, parasomnias of Rapid Eye Movement (REM) sleep, sleep-wake transition disorders and nonspecific parasomnias (Davis, Parker, & Montgomery, 2004; Thorpy, 2012).

Within the scope of this thesis, it is not possible to describe all sleep disorders occurring for children with ASD. Dyssomnias will be discussed within this thesis as parasomnias are outside of the thesis scope and the extant literature has primarily focused on the presence of dyssomnias in both TD children and children with developmental disabilities (Liu, Hubbard, Fabes, & Adam, 2006; Malow, 2004; Wiggs, 2001).

Co-sleeping. Co-sleeping is defined as either one or both parents sharing a bed or being asleep in the same sleeping space as the child (Dodd & Jackiewicz, 2015; McKenna & Volpe, 2007). This can be intentional or reactive (Dodd & Jackiewicz, 2015). Intentional co-sleepers are families/parents who intentionally share their beds with their children because they believe it to be the best sleep arrangement for their family (Goldberg & Keller, 2007). Whereas reactive co-sleeping is where families report difficulties with problematic sleep behaviours and allow co-sleeping, even though parents would prefer separate sleeping arrangements (Goldberg & Keller, 2007). Co-sleeping is also highly dependent on cultural influences (Huang, Wang, Zhang, & Liu, 2010; Li et al., 2009). Due to social values and differences in socio-economic status, there are large cross-cultural variations in regard to the acceptability of co-sleeping (Huang et al., 2010; Li et al., 2009).

Research has demonstrated that poor lifestyle and sleep practice are associated with child sleep disturbances especially co-sleeping in children with ASD. These include stimulating activities before bedtime, poor sleeping arrangements, poor parenting skills, children without their own bed or room, late and inconsistent bedtime and a lack of exercise during the day (Huang et al., 2010; Liu et al., 2006). Three factors may account for the high percentage of co-sleeping in children with ASD. Firstly, because of their disability, parents may think that their child may need more care during the night than a TD child (Liu et al., 2006). Secondly, children's repetitive and restricted behaviours may contribute to the likelihood for co-sleeping with parents and it may be difficult to separate the child into a new sleeping environment (Liu et al., 2006). Thirdly, to compensate for their child's bedtime resistance, parents may allow co-sleeping in children with ASD because of their poor night-time settling or anxieties around going to bed (Cotton & Richdale, 2006). However, co-sleeping can become problematic because it may lead to settling difficulties and conditional night-time awakenings (Ferber & Kryger, 1995).

Prevalence of Sleep Problems in ASD

According to previous studies, the prevalence of sleep problems in children with ASD has been reported to range between 40 to 80% (Angriman, Caravale, Novelli, Ferri, & Bruni, 2015; Doo & Wing, 2006; Goldman et al., 2011; Hoffman et al., 2005; Johnson, 1996; Köse, Yılmaz, Ocakoğlu, & Özbaran, 2017; Krakowiak et al., 2008; Liu et al., 2006; Malow et al., 2006; Mannion, Leader, & Healy, 2013a; Park et al., 2012; Polimeni, Richdale, & Francis, 2005; Richdale, 1999; Schreck & Mulick, 2000; Souders et al., 2009; Wiggs, 2001). This compares to 10%-35% for typically developing children (TD) (Armstrong, Quinn, & Dadds, 1994; Byars, Yolton, Rausch, Lanphear, & Beebe, 2012; Couturier et al., 2005; Davis et al., 2004; Gaylor, Goodlin-Jones, & Anders, 2001; Krakowiak et al., 2008; Lam, Hiscock, & Wake, 2003; Owens & Moore, 2017; Richdale & Schreck, 2009a; Scher et al., 1995; Singh & Zimmerman, 2015; Wake et al., 2006), 25%-50% for children with ADHD (Corkum, Tannock, & Moldofsky, 1998; Meltzer & Mindell, 2008; Spruyt & Gozal, 2011), and 24% to 68% for children with other developmental or intellectual disabilities (Carter, McCaughey, Annaz, & Hill, 2009; de Miguel-Díez, Villa-Asensi, & Álvarez-Sala, 2003; Didden, Korzilius, Aperlo, Overloop, & Vries, 2002; Didden & Sigafos, 2001; Krakowiak et al., 2008; Robinson & Richdale, 2004; Stores & Stores, 2013; Stores & Stores, 2004; Wiggs & Stores, 1996).

It is difficult to get an accurate measure of sleep problems in children with ASD due to parents over-reporting their child's sleep difficulties, the different methodology used in studies and both environmental and cultural factors (Goodlin-Jones, Tang, Liu, & Anders, 2008; Hering, Epstein, Elroy, Iancu, & Zelnik, 1999). In spite of this variability, there is a large body of research to indicate that children with ASD are particularly vulnerable to sleep disturbances (Couturier et al., 2005; Krakowiak et al., 2008; Richdale & Prior, 1995).

Trajectory of Sleep Problems

If not effectively treated, sleep difficulties in children with ASD commonly last for many years and can persist into adulthood (Baker & Richdale, 2015; Cohen, Conduit, et al., 2014; Goldman, Richdale, Clemons, & Malow, 2012; Matson, Ancona, & Wilkins, 2008; Robinson & Richdale, 2004). Evidence suggests the persistence of sleep problems in children with ASD is around 63% (Cortesi, Giannotti, Ivanenko, & Johnson, 2010), while some sleep difficulties have shown to increase over time from 84% to 87.5% (Cortesi, Giannotti, Ivanenko, & Johnson, 2010; Hodge, Carollo, Lewin, Hoffman, & Sweeney, 2014). Sleep problems have also been found to persist for long periods in children with other developmental disabilities (e.g., Rett syndrome) and children with intellectual disability (Polimeni et al., 2005; Quine, 1991; Richdale, Francis, Gavidia-Payne, & Cotton, 2000; Wong, Leonard, Jacoby, Ellaway, & Downs, 2015). This is in contrast to research with TD children which suggests that sleep problems in TD children decline with age (e.g., 72% to 37.5% from age 2-5 to 10-17 years old) (Allik, Larsson, & Smedje, 2008; Gregory & O'Connor, 2002; Hodge, Carollo, Lewin, Hoffman, & Sweeney, 2014; Richdale & Prior, 1995; Sivertsen, Posserud, Gillberg, Lundervold, & Hysing, 2012). As sleep problems are unlikely to abate without effective treatment, it is essential that we identify such treatments for children with ASD.

Causes of Sleep Problems in Children with ASD

There are differing theories about the reasons for higher rates of sleep disturbances in children with ASD. Often TD children experience sleep problems because they do not follow a bedtime routine, they lack self-soothing skills, poor parental limit setting, shared sleeping arrangements, crowded housing, noise and medical conditions (Adair, Bauchner, Philipp, Levenson, & Zuckerman, 1991; Anders, Halpern, & Hua, 1992; Bonuck & Grant, 2012; Sadeh, 2005; Sadeh, Mindell, Luedtke, & Wiegand, 2009). However, the etiologies for sleep

problems in children with ASD are thought to be multifactorial with environmental, genetic, social, biological, psychological and behavioural factors thought to play a role (Brown, 2014; Cortesi, Giannotti, Ivanenko, & Johnson, 2010; Richdale, 2013).

Environmental factors. Adaptive sleep habits and routines commonly referred to as “sleep hygiene” can improve sleep quality and reduce difficulties with getting to sleep by addressing poor sleep habits in individual children and promoting appropriate sleep parameters (Spruyt & Curfs, 2015). Although poor sleep hygiene may not be the sole cause of sleep problems in children with ASD, it can perpetuate sleep difficulties (Sharma & Andrade, 2012). It is important for sleep hygiene to be addressed otherwise other sleep interventions will likely be unsuccessful (Johnson, Giannotti, & Cortesi, 2009).

There are a number of variables relating to sleep hygiene that may contribute toward sleep problems in children with ASD. These include the temperature of the bedroom, position of the bed, bedding, sleep clothes, noise levels, light, visual stimuli, crowded housing as well as colour and movement of an object in the bedroom (Devnani & Hegde, 2015; Kohlhuber & Bolte, 2011; Liu et al., 2006; Simakajornboon, Kheirandish-Gozal, & Gozal, 2009). For children with ASD sleep may be a more effortful process than for TD children, possibly because they need to disengage from the sensory environment around them. In addition, sensitivity to the environment has been shown to increase psychological arousal and release of cortisol which is likely to impede on sleep (Reynolds & Malow, 2011; Scher, Hall, Zaidman-Zait, & Weinberg, 2010; Schoen, Miller, Brett-Green, & Nielsen, 2009). Therefore, it is important that the optimal sleep environment is provided.

A behavioural model of sleep disturbance. Sleep can best be viewed as a biobehavioural state in which a combination of biological and behavioural variables impact upon the initiation of sleep and sleep maintenance (Blampied & France, 1993; Didden et al.,

2002). Behaviour theory specifies that antecedent events and contingencies of reinforcement can impact the probability of future occurrences of that behaviour (Blampied, 2013; Skinner, 1969). Sleep itself is not an operant behaviour as it is not possible to increase or decrease the frequency or duration through contingencies of reinforcement or punishment. However, behaviour that occurs as part of the pre-bedtime routine and the transition into falling asleep is a part of a complex behaviour chain that is reinforced by the event of sleep itself (Blampied, 2013; France & Blampied, 2005). Behaviour that is reinforced by sleep comes to operate under the control of discriminative stimuli that are present in the environment at the time of reinforcement (Blampied, 2013; Skinner, 1969). Stimuli that are present in the sleep environment such as comfortable bedding, cool temperature and a quiet and dark room are common discriminative stimuli that affect the likelihood of sleep (Blampied, 2013).

If a parent is regularly present in the bedroom prior to the child falling asleep (e.g., co-sleeping), then the presence of the parent becomes a discriminative stimulus for sleep and will likely be required for the child to return to sleep if they wake during the night (Blampied & France, 1993; France & Blampied, 1999). This can become problematic when these conditions are undesired (i.e., when the goal is to achieve independent sleep initiation). Contingencies of reinforcement can also play a significant role in sleep problems such that parental attention becomes positive reinforcement for the child during the night.

Changing these patterns of behaviour can require an assessment of the unique antecedents and consequences maintaining the behaviour and the development of interventions which address these unique variables. In order to eliminate unwanted behaviours that interfere with a child's ability to fall asleep, it is important that the environmental conditions, discriminative stimuli, and consequence variables are examined so that appropriate natural cues become the controlling stimulus for sleep and appropriate sleep-related behaviours are reinforced (Blampied, 2013).

Familial factors. Familial factors associated with the development and maintenance of sleep problems in children with ASD include poor maternal health, a family history of sleep disorders, ill siblings, parental stress, poor parenting skills, inconsistent or late bedtimes and marital discord (Bell & Belsky, 2008; Meltzer & Mindell, 2007; Richdale & Schreck, 2009a; Stores, 1996). For example, Thunstrom and Ferber (1999) found stress and feelings of incompetence, as well as restriction concerning the parental role, were associated with frequent night waking and prolonged sleep latency reported by parents of more than 2,500 children aged from six to 12 months. Another study by Sadeh, Raviv, and Gruber (2000) linked family stress to sleep problems among older children aged seven, nine and 11 years of age. They found that the best predictor of sleep quality proved to be family stress including emotionally arousing events such as hospitalisations, illnesses and general emotional turmoil (Sadeh et al., 2000).

Psychological/behavioural characteristics. Children with ASD experience high rates of externalising and internalising problems (Gillott et al., 2001; Kuusikko et al., 2008; Lainhart, 1999; Magnuson & Constantino, 2011; Mazefsky, Conner, & Oswald, 2010; Russell & Sofronoff, 2005; White et al., 2009). Externalising behaviours such as hyperactivity, aggression and disruptive behaviour have been linked with sleep difficulties in children with ASD (Dahl & Harvey, 2007; Hollway & Aman, 2011; Mayes & Calhoun, 2009; Meltzer & Mindell, 2008). Furthermore, previous research suggests that internalising disorders such as anxiety and depression may independently or in combination with other factors contribute toward sleep problems in children with ASD. For example, children with ASD and anxiety may have trouble falling asleep due to increased autonomic activity (e.g., heart rate and cortisol levels) and psychological arousal (Mazurek & Petroski, 2015; Tani et al., 2004). Furthermore anxiety may be triggered by environmental factors and become intensified by ASD symptoms contributing to sleep difficulties (Hollway & Aman, 2011;

May, Cornish, Conduit, Rajaratnam, & Rinehart, 2015) It is important to note that the directionality of the relation between internalising problems and sleep is unclear because of the overlap between these problems and the core ASD symptoms (May et al., 2015; Sukhodolsky et al., 2008).

It is possible that a cognitive process can play a role in sleep problems in children with ASD as hypothesised by Richdale, Baker, Short, and Gradisar (2014). They put forward the idea that that intrusive thoughts and worries about sleep during the pre-sleep period produce emotional distress and increased psychological arousal resulting in continued cognitive activity leading to interference with sleep onset (Harvey, 2002; Mazurek & Petroski, 2015).

Diagnostic features of ASD. Sleep difficulties in children with ASD may be related to associated core features of ASD such as delayed development and social-communication deficits. In addition to the light-dark cycle, humans use social cues to entrain circadian rhythms. Routines and social cues are thought to be important to help children develop stable sleep-wake patterns with the longest sleep occurring during the night. Therefore, children with a social-communication deficit may find it difficult to use such cues to entrain their rhythms resulting in problems with their sleep-wake schedule (Johnson, 1996; Richdale & Prior, 1995). For example, communication difficulties reported on the GARS (Gilliam Autism Rating Scale) have been found to predict sensitivity to the sleeping environment (Richdale & Schreck, 2009a). Additionally, due to communication deficits, children may not understand the parent's instructions and expectations around going to bed and falling asleep (Malow et al., 2014). One other common behavioural manifestation of children with ASD is an attachment to routine or rituals. The non-functional routines common in children with ASD may result in settling difficulties and bedtime resistance, especially if these routines are not able to be met (Richdale, 1992; Richdale, 1999). Lastly, children with ASD often have

trouble with emotional regulation (e.g., ability to calm oneself) and transitioning from stimulating or preferred activities to sleep, this may impede the establishment of consistent bedtime routines and behaviours (Malow et al., 2014).

Allowing the child to engage in repetitive sensory-motor behaviours earlier in the evening (outside of their bedroom) and engaging in calming activities an hour before bedtime may be useful strategies to reduce ongoing sleep problems associated with diagnostic features of ASD (Hundley et al., 2016; Owens, 2017).

Preliminary evidence suggests that increased ASD symptom severity may predict an increased probability of the presence of sleep problems (Adams et al., 2014; Hoffman et al., 2005; Park et al., 2012; Tudor et al., 2012). For example, insufficient sleep including short sleep duration and sleep onset delay has been known to exacerbate the severity of core ASD symptoms (e.g., social and communication difficulties and repetitive behaviours) as well as overall ASD severity (Schreck, Mulick, & Smith, 2004; Tudor et al., 2012). Furthermore, research has linked specific types of repetitive stereotypic behaviour with sleep problems in ASD (Hundley et al., 2016; Schreck et al., 2004). For example, higher rates of repetitive sensory-motor movements were correlated with shortened sleep duration and screaming during the night (Hundley et al., 2016; Schreck et al., 2004).

Moreover, children with ASD who were poor sleepers were rated by their parents as having more compulsive and ritualistic behaviours compared to those defined as good sleepers (Goldman et al., 2009; Hundley et al., 2016). Higher rates of restricted and compulsive behaviours, as well as the need for sameness, were correlated with increased night-time fragmentation of sleep for both poor sleepers and good sleepers with ASD (Goldman et al., 2009).

Melatonin levels and circadian rhythms. Melatonin is a neurohormone which is responsible for the regulation of the circadian sleep-wake rhythm (Arendt, 2005; Didden & Sigafos, 2001; Doyen et al., 2011; Tordjman et al., 2013). A collection of studies suggest that neurodevelopmental disorders predispose children to sleep-wake rhythm disturbances (Cohen, Conduit, et al., 2014; Jan & O'Donnell, 1996; Richdale & Schreck, 2009b; Sajith & Clarke, 2007). Research has identified abnormally low levels of melatonin in children with ASD as well as abnormalities regulating basic circadian rhythms. This may make it difficult for individuals with ASD to establish a 24-hour sleep-wake cycle (Jan, Freeman, & Fast, 1999; Kulman et al., 2000; Melke et al., 2008; Tordjman et al., 2013). Specifically, difficulties related to sleep maintenance, sleep onset, total sleep time as well as nocturnal and early morning awakenings in children with ASD (Cortesi, Giannotti, Sebastiani, Panunzi, & Valente, 2012; Giannotti, Cortesi, Cerquiglini, & Bernabei, 2006; Patzold, Richdale, & Tonge, 1998; Richdale, 1999; Richdale, 2001; Tordjman et al., 2013).

Co-morbidities/medical issues. Children with ASD have an increased likelihood of co-occurring comorbidities that may affect sleep such as seizure disorders, ADHD, restless leg syndrome, sleep apnoea, rhythmical movement disorder and psychiatric disorders (Dillon & Chervin, 2014; Leyfer et al., 2006; Maski, Jeste, & Spence, 2011; Rodriguez, 2007). These comorbidities can be associated with settling difficulties, sleep onset delay due to repetitive motions of the head, trunk or limbs, hyperarousal, elevated moods, heightened anxiety around bedtime, a decrease in sleep efficiency due to night wakings and early morning wakings (Hoban, 2003; Richdale & Schreck, 2009).

Further to this, many children with ASD are on medication, such as clonidine, diphenhydramine, psychotropics, and antiepileptics to treat psychopathology or medical conditions and these can significantly disrupt children's sleep (e.g., the effect of stimulant medication for ADHD including both the direct alerting effects and the hyperactivity

associated with withdrawal) (Aman, Lam, & Collier-Crespin, 2003; Bazil, 2003; Liu et al., 2006; Malow, Byars, et al., 2012; Mayes & Calhoun, 2009; Singh & Zimmerman, 2015).

Other medical conditions that are common in children with ASD and that are associated with sleep difficulties include milk allergies, gastrointestinal problems, asthma, sleep-related headaches that occur during sleep or upon awakening, upper respiratory infections, and several types of sleep-related epilepsy (e.g., benign epilepsy of childhood with centrotemporal spikes) (American Psychiatric Association, 2013; Bisulli et al., 2012; Camhi, Morgan, Pernisco, & Quan, 2000; Liu et al., 2006; Mannion, Leader, & Healy, 2013b; Owens & Witmans, 2004). Based on research in this area, it appears that sleep problems in children with ASD are likely to be the result of interactions of multiple genetic, medical, neurodevelopmental and environmental factors (Liu et al., 2006; Richdale, 1999). The extent to which these factors underpin sleep problems for each child is variable, but must all be considered in formulating treatments for sleep problems in children with ASD.

Common Sleep Treatments

Pharmacological treatments. A number of treatments have been developed to overcome sleep problems in children with ASD. These include medications as well as behavioural approaches. Melatonin supplementation is a common medical treatment and has shown to be effective at improving sleep initiation with minimal adverse side effects (Braam et al., 2009; Malow et al., 2012). Melatonin is an endogenous hormone involved in the body's regulation of sleep. Melatonin can be used as a chronobiotic to shift or advance the timing of sleep onset. Melatonin is usually given in lower doses 3 to 5 hours before bedtime. Once a sleep cycle is established for 6 weeks or more, the melatonin may be discontinued. However, long-term use is often necessary to maintain sleep patterns (Giannotti, Cortesi, Cerquiglioni, & Bernabei, 2006). Other pharmacological treatments that have been used to treat sleep disturbance in children with ASD include mirtazapine (Posey, Guenin, Kohn, Swiezy, &

McDougle, 2001), clonidine (Ming, Gordon, Kang, & Wagner, 2008) and gabapentin (Robinson & Malow, 2013). These psychotropic medications have been evaluated to treat insomnia in children with ASD effectively (Malow et al., 2012).

Sleep hygiene. Usually, the initial step in treating sleep difficulties in children with ASD is to improve sleep hygiene defined as sleep-related behaviours that promote effective and properly timed sleep (Blackmer & Feinstein, 2016; Jan et al., 2008). Sleep hygiene facilitates good sleep quality, reduces sleepiness during the day and can reduce or eliminate sleep problems (Lebourgeois, Giannotti, Cortesi, Wolfson, & Harsh, 2005; Mindell, Meltzer, Carskadon, Chervin, 2009; Van Der Heijden, Stoffelsen, Popma, & Swaab, 2018). Bedtime routines are a fundamental component of promoting good sleep, they are easy to implement and have few reported negative side effects (Kodak & Piazza, 2008).

Sleep hygiene practices cover a number of domains including the sleep routine (e.g., creating a calming and structured bedtime routine), scheduling (e.g., appropriate sleep times with adequate sleep opportunity depending on age), the sleep environment (e.g., reducing stimuli before bed, temperature, ambient light), and daytime activities (e.g., caffeine use, exercise, adjusting the timing of meals) (Cavalieri, 2016; Jan et al., 2008; Mindell, Meltzer, Carskadon, & Chervin, 2009a; Moore, 2012; Vriend et al., 2011). A good bedtime routine can prepare a child to sleep (Devnani & Hegde, 2015). The evidence is clear that without proper sleep hygiene practices, sleep patterns often diverge from developmentally appropriate norms (Jan et al., 2008). Additionally, if poor sleep habits are unrecognised or positive sleep hygiene practices are not put in place, other behavioural interventions will likely be unsuccessful (Jan et al., 2008). Implementing good sleep hygiene is an important contributor to sleep quality across the lifespan (Mindell, Meltzer, Carskadon, & Chervin, 2009; Tan, Healey, Gray, & Galland, 2012).

Research has shown that sleep hygiene can reduce or eliminate sleep problems and improve sleep quality across the lifespan and contribute to successful management of sleep problems in ASD (Brown, 2014; Singh & Zimmerman, 2015; Spruyt & Curfs, 2015). Good sleep hygiene involves not only good bedtime routines but also includes good habits during the daytime as well as establishing an optimal sleeping environment (Singh & Zimmerman, 2015). For example, limiting caffeine intake, limiting daytime naps and decreasing visual stimulation from technology (Jan et al., 2008; Malow et al., 2014; Singh & Zimmerman, 2015).

There is little research investigating the impact of sleep hygiene in the pediatric population. Research into the effects of modifications to sleep hygiene on sleep have typically focused on children with developmental disabilities (Mindell, Meltzer, Carskadon, & Chervin, 2009). Recommendations around sleep hygiene are mostly incorporated into behavioural sleep programmes, but have rarely been evaluated independently of a sleep programme (Galland & Mitchell, 2010).

Sleep hygiene has proved to be effective independently (LeBourgeois, Giannotti, Cortesi, Wolfson, & Harsh, 2005) and in combination with other behavioural interventions to address sleep problems in TD children (LeBourgeois et al., 2005; Mindell, Leichman, Lee, Williamson, & Walters, 2017; Mindell, Li, Sadeh, Kwon, & Goh, 2015; Mindell, Meltzer, et al., 2009a; Mindell, Telofski, Wiegand, & Kurtz, 2009; Vriend et al., 2011). Research with TD children suggests that sleep hygiene and a consistent bedtime routine has shown to be associated with better sleep outcomes including shorter sleep onset latency, increased sleep duration and earlier bedtimes (Mindell et al., 2017; Mindell et al., 2015; Moore, 2012).

Five studies were found that have investigated sleep hygiene in children with ASD in combination with other behavioural treatments (Durand & Christodulu, 2004; Durand,

Gernert-Dott, & Mapstone, 1996; Montgomery, Stores, & Wiggs, 2004; Moon, Corkum, & Smith, 2011; Reed et al., 2009). Four studies investigated the effectiveness of sleep hygiene separately from other sleep treatments in children with ASD (Adkins et al., 2012; Malow et al., 2014; Piazza, Fisher, & Sherer, 1997; Weiskop, Richdale, & Mathews, 2005). The outcomes of these four studies were inconsistent. Two studies found that sleep hygiene alone did not eliminate sleep problems in children with ASD (Adkins et al., 2012; Weiskop et al., 2005) whilst the others found that sleep hygiene slightly improved sleep problems in one child, however, the other participant showed no improvement (Piazza et al., 1997). Additionally, sleep latency, night wakings and sleep duration were shown to slightly improve after promoting healthy bedtime routines (Malow et al., 2014). The successful effects of sleep hygiene are hard to be determined if combined with other behavioural treatments (Christodulu & Durand, 2004; Vriend et al., 2011; Weiskop et al., 2005).

Behaviourally Based Treatments for Sleep Problems in Children with ASD and the Evidence of Effectiveness in both TD Children and Children with ASD.

Behavioural approaches to the treatment of sleep problems in children with ASD include scheduled awakenings, bedtime fading, sleep restriction, standard extinction and modifications to extinction, such as graduated extinction (Richdale & Wiggs, 2005; Turner & Johnson, 2013; Vriend et al., 2011). The purpose of behaviourally based interventions is to decrease undesirable behaviours and encourage appropriate behaviours. Behavioural interventions involve identifying factors that reinforce the problem behaviour (e.g., technology in the bedroom) and replacing or withdrawing these (Jones & Verduyn, 1983; Weiskop, Matthews, & Richdale, 2001). Intervention does not involve changing the child's sleep per se, but rather changing the parent's attitudes and behaviours towards the child at bedtime and during sleep, which in turn alters the child's behaviour (France, Henderson, & Hudson, 1996). Behaviourally based interventions are based on the behavioural model of

insomnia and the principles of learning theory to bring about change in how one responds to certain events or stimuli (Meltzer & Mindell, 2014; Owens, France, & Wiggs, 1999; Weiskop et al., 2001). Pure behavioural interventions also include a cognitive component, especially, working with parents to alter beliefs, thoughts, behaviours and attitudes toward their child's sleep problems so they are inclined to engage in a behaviorally based sleep intervention programme (Owens et al., 1999). Behavioural interventions are viewed as an adequate alternative to pharmacological interventions and are often a treatment of choice for paediatric sleep problems (Owens et al., 1999).

Behavioural treatments for sleep problems have a large number of advantages over pharmacological treatments (Vriend et al., 2011). These include an increased sense of parental competence and control (Vriend et al., 2011; Wolfson, Lacks, & Futterman, 1992), parental preference with behavioural interventions over sleep-enhancing medications (Vriend et al., 2011; Williams, Sears, & Allard, 2004) and generally there are long-lasting benefits with fewer side effects (Christodulu & Durand, 2004). The research to each of these approaches is described below.

Scheduled awakenings. A scheduled awakening procedure involves the parents waking their child before the time when the child usually spontaneously wakes (Durand & Mindell, 1999; Mindell et al., 2006; Morgenthaler et al., 2006; Vriend & Corkum, 2011). Over time, the waking time is extended so that this occurs following longer intervals of sleep, and are gradually faded out (Turner & Johnson, 2013). Research with TD children suggests scheduled awakenings are effective in treating spontaneous night-time awakening and crying episodes (Johnson & Lerner, 1985; Rickert & Johnson, 1988). One study has investigated a scheduled awakening procedure in children with ASD (Piazza et al., 1997). The finding of this study suggests that scheduled awakenings are an effective treatment to increase total

sleep time. This intervention meets the requirements of being possibly efficacious (Vriend et al., 2011).

Faded bedtime. A faded bedtime procedure involves determining a time, which is likely for the child to fall asleep within 15 minutes of going to bed (Vriend et al., 2011). Once the child reliably falls asleep at this time, the bedtime is gradually modified to an earlier bedtime until the child falls asleep at an acceptable time of night (Schreck, 2001).

Additionally, the child is awakened at the same time each morning and is not allowed to nap outside of the scheduled sleep time (Piazza et al., 1997; Vriend et al., 2011). Research with TD children suggests faded bedtime is an effective treatment to improve sleep latency, number of awakenings and waking after sleep onset (Ashbaugh & Peck, 1998; Piazza & Fisher, 1991). Three studies have investigated faded bedtime in children with ASD (DeLeon, Fisher, & Marhefka, 2004; Moon, Corkum, & Smith, 2011; Piazza, Fisher, & Sherer, 1997). The findings of these studies suggest that faded bedtime reduces disturbed sleep, nighttime awakenings, night waking SIB, sleep onset latency and shows improvements in daytime behaviour with improvements maintained at three months (Moon et al., 2011) and four months (Piazza et al., 1997) following treatment. However, faded bedtime does not meet criteria to be possibly efficacious due to a lack of methodological rigour (Vriend et al., 2011).

Faded bedtime is usually a multicomponent intervention package with response cost. It is unknown whether both components of the package are necessary to achieve positive results. Further research on individual components on bedtime fading should be conducted (Ashbaugh & Peck, 1998).

Sleep restriction. Sleep restriction involves temporarily reducing the amount of time that a child spends in bed to only 90% of the total amount of time they usually sleep. Once behaviour improves, the sleep schedule is gradually altered to better resemble what is

recommended for the child's age (Turner & Johnson, 2013). These strategies have been shown to decrease target sleep problems for the majority of participants, and are seen as socially acceptable to parents (Turner & Johnson, 2013).

Research with TD children is scarce but suggests that sleep restriction improves night waking^s and improves total sleep time (Sadeh, Gruber, & Raviv, 2003). The efficacy of sleep restriction as a stand-alone intervention in TD children has yet to be comprehensively reviewed (Miller et al., 2014).

Two studies have investigated sleep restriction in children with ASD (Christodulu & Durand, 2004; Durand & Christodulu, 2004). The findings of these studies suggest that sleep restriction is effective in reducing co-sleeping, night waking, sleep association problems, bedtime resistance, hours of disturbed sleep and increased parental satisfaction with their child's behaviour at bedtime (e.g., time it took to put child to bed, current sleep pattern) (Christodulu & Durand, 2004; Durand & Christodulu, 2004).

Standard extinction. Standard extinction refers to a traditional extinction process e.g., planned ignoring and systematic ignoring whereby parents ignore all bedtime disruptions and nighttime waking including screams, cries and tantrums (Vriend et al., 2011).

Research with TD children suggests that extinction is highly effective in reducing bedtime problems, nighttime awakenings and improving sleep time continuity (Chadez & Nurius, 1987; Didden, De Moor, & Kruit, 1999; France, Blampied, & Wilkinson, 1991; Friman, 2000; Reid, Walter, & O'Leary, 1999; Rickert & Johnson, 1988; Roberts, 1993; Ronen, 1991; Seymour, Brock, During, & Poole, 1989; Williams, 1959; Wright, Woodcock, & Scott, 1970). However, much of this literature is only marginally relevant in the current review because it considers infants and not young children.

Four studies have investigated standard extinction in children with ASD (Weiskop et al., 2001; Weiskop et al., 2005; Wolf, Risley, Johnston, Harris, & Allen, 1967; Wolf, Risley, & Mees, 1963). The findings of these studies suggest that standard extinction is effective in improving night-time awakenings, self-settling and reducing co-sleeping. Improvements in night-time awakenings were maintained at either six month (Wolf et al., 1967) or 12 month (Weiskop et al., 2001; Weiskop et al., 2005) follow-ups. Standard extinction meets criteria for a possibly efficacious intervention (Vriend et al., 2011). However, strong extinction bursts are often seen when using this intervention. This can be stressful for parents and is, therefore, less socially valid (Ferber, 1985; Schreck, 2001).

Graduated extinction. Graduated extinction involves gradually removing reinforcement to reduce behaviour (Vriend et al., 2011). In this procedure, parents follow a systematic routine and put the child to bed. If the child screams, cries or tantrums during the night, the parents initially ignore this behaviour for a pre-set time period (typically five minutes). If the child continues to cry at the end of that time period, the parent returns to the room and settles the child with as little attention as possible. The parent then leaves the room (Turner & Johnson, 2013). These approaches have been used successfully for children with developmental disabilities including children with ASD (Vriend et al., 2011).

Research with TD children suggests that graduated extinction is effective in reducing bedtime problems and night-time awakenings (Adams & Rickert, 1989; Durand & Mindell, 1990; Eckerberg, 2002; Fisher, Feekery, & Rowe, 2004; Hiscock & Wake, 2002; Lawton, France, & Blampied, 1991; Leeson, Barbour, Romaniuk, & Warr, 1994; Minde, Faucon, & Falkner, 1994; Mindell & Durand, 1993; Pritchard & Appleton, 1988; Reid, Walter, & O'Leary, 1999; Roberts, 1993; Rolider & Houten, 1984; Sadeh, Gruber, & Raviv, 2002).

Three studies have investigated graduated extinction in children with ASD (Durand et al., 1996; Montgomery et al., 2004; Moore, 2004). The findings of these studies suggest that

graduated extinction reduces sleep onset latency, co-sleeping, bedtime disturbance and bedtime resistance. More methodically rigorous research examining the effectiveness of graduated extinction is needed for it to be labelled as possibly efficacious (Vriend et al., 2011).

To summarise, behavioural interventions are highly effective in improving sleep problems in TD children, but relatively few published studies have examined behavioural treatments of sleep problems in children with ASD, and limited research has been conducted to determine the effectiveness of these behavioural interventions for children with ASD. It appears that there is no single intervention that is effective across all components of sleep problems in individuals with ASD (Cortesi, Giannotti, Sebastiani, Panunzi, & Valente, 2012; Malow et al., 2014; Reed et al., 2009).

A previous review of the literature has demonstrated strong empirical evidence for the efficacy of these treatments described, however, no intervention meets criteria to be classified as well-established (Schreck, 2001). Only standard extinction has met criteria to be considered possibly efficacious for children with ASD (Schreck, 2001; Turner & Johnson, 2013). Although standard extinction is seen as effective, it may not be socially valid for some families (Turner & Johnson, 2013). At this point, there has not been enough research investigating other behavioural strategies such as graduated extinction and faded bedtime to allow firm conclusions of treatment effectiveness for individuals with ASD (Vriend et al., 2011). For treatments to be well-established such treatments and studies need replication. Many more studies are needed with sound methodological designs to be considered possibly efficacious (Vriend et al., 2011).

The research has demonstrated the short to medium term effectiveness of behavioural sleep interventions but due to the limited research, little is known about the sustainability of

changes over the longer term (Ospina et al., 2008). In the absence of longer-term follow-up studies within the ASD population, it is entirely possible that benefits may extend beyond the follow-up studies that have been already demonstrated (Hiscock, Bayer, Hampton, Ukoumunne, & Wake, 2008).

Secondary Effects of Sleep Disturbance in Children with ASD

Child outcomes. An extensive body of literature has shown that sleep problems can have a profound impact on children. Emerging evidence suggests sleep problems can exacerbate the difficulties children with ASD have with social interactions, communication skills, routines and stereotypy (Park et al., 2012; Taylor et al., 2012; Tudor et al., 2012; Goldman et al., 2011; Lambert, 2016; May, Cornish, Conduit, Rajaratnam, & Rinehart, 2015; Richdale & Schreck, 2009; Schreck et al., 2004; Taylor, Schreck, & Mulick, 2012; Goldman et al., 2011; Reed et al., 2009; Schreck et al., 2004; Tudor, Hoffman, & Sweeney, 2012; Vriend et al., 2011). In addition, sleep difficulties have been associated with increased rates of daytime behaviour problems, physical health problems, internalising and externalising behaviour problems, cognitive deficiencies, and overall severity of ASD (Goldman et al., 2011; Hollway et al., 2013; Liu et al., 2006; Malow et al., 2006; Paavonen, Nieminen-von Wendt, Vanhala, Aronen, & Von Wendt, 2003; Schreck et al., 2004; Schwichtenberg et al., 2013; Williams, Sears, & Allard, 2004). It is difficult to determine the extent to which sleep disturbance may exacerbate these problems (Karst & Van Hecke, 2012). Therefore, examining these associations in children with ASD is of vital importance.

Externalising behaviour. Externalising behaviours can include aggression, hyperactivity, rule-breaking, impulsivity and control problems (Bauminger et al., 2010). Researchers from a number of studies have examined the association between externalising behaviours and sleep disturbance in children with ASD (Allik, Larsson, & Smedje, 2006; Bruni et al., 2007; Dahl & Harvey, 2007; DeVincent, Gadow, Delosh, & Geller, 2007;

Goldman et al., 2009; Henderson, Barry, Bader, & Jordan, 2011; Malow et al., 2006; Mayes & Calhoun, 2009; Moon et al., 2011; Patzold et al., 1998; Sikora, Johnson, Clemons, & Katz, 2012). However, findings are inconsistent, especially in regard to parent and teacher reports of externalising behaviours.

For example, DeVincent et al. (2007) assessed 112 preschool children with PDD and found that those who had sleep problems exhibited more externalising behaviour such as severe symptoms of oppositional defiant disorder, attention-deficit hyperactivity disorder and hypersensitivity to stimulus compared to children without sleep problems (DeVincent et al., 2007; Liu et al., 2006). Similarly, Bruni et al. (2007) found an inverse association between percentage of sleep efficiency and the CBCL externalising behaviour composite scores. Higher composite scores are associated with lower percentage of sleep efficiency. Furthermore, both, Henderson et al. (2011) and Sikora et al. (2012) found that sleep quality and sleep hygiene were significantly negatively correlated with externalising behaviours.

Goldman et al. (2011) investigated the relationship between sleep and daytime behaviour in 1784 children with ASD. Sleep problems were identified using the CSHQ. Behavioural problems among children rated as being either good or poor sleepers were evaluated using the Parental Concerns Questionnaire (PCQ). Goldman et al. (2011) found that if the parent reported their child to be a poor sleeper, there was a higher risk of having problematic daytime behaviours on all PCQ scales (e.g., compulsive behaviour, aggression, hyperactivity, mood swings, self-stimulatory behaviour and self-injurious behaviour). Furthermore, children with ASD who were classified as poor sleepers by parent report on the PCQ had higher scores on the CBCL. While this study has limitations in terms of its cross-sectional design and lack of objective measurements of sleep and behaviour, these findings are consistent with previous reports on the association of sleep and challenging daytime behaviour in children with ASD (DeVincent et al., 2007; Goldman et al., 2009; Goodlin-

Jones, Tang, Liu, & Anders, 2009; Liu et al., 2006; Malow et al., 2006; Mayes & Calhoun, 2009).

In contrast to the aforementioned findings, Goodlin-Jones et al. (2009) found that no daytime behaviour measures on the CBCL were associated with Research Diagnostic Criteria (RDC) insomnias. Specifically, no significant associations were found between sleepiness and daytime behaviour (Goodlin-Jones et al., 2009).

A few studies have specifically examined the relationship between sleep disturbance and hyperactivity/inattention (Allik et al., 2006; Goldman et al., 2009; Malow et al., 2006; Mayes & Calhoun, 2009). The findings of these studies are mixed. For example, Goldman et al. (2009) used the PCQ to assess hyperactivity and inattentiveness in their study sample (e.g., children with ASD who were good sleepers, poor sleepers and TD children who were good sleepers). Parents of children with ASD who were poor sleepers reported more inattention and hyperactivity in their children on the PCQ than parents of children who were good sleepers.

By contrast, Malow et al. (2006) found that parents of children with ASD who were poor sleepers reported no more hyperactivity and inattention in their children than those who were good sleepers. Additionally, Mayes and Calhoun (2009) found that the sleep subscale scores on the Paediatric Behaviour Scale (PBS) were related to the attention deficit and hyperactivity subscale scores on the PBS. However, teacher ratings of hyperactivity and inattentiveness did not support this finding and were not related to the sleep subscale score on the PBS (Mayes & Calhoun, 2009).

Few studies have investigated the effects of resolving sleep problems on children's problem behaviour. The research that exists suggests that following behavioural interventions improvements were found in sleep disturbance and daytime problem behaviour in children

with ASD. For example, Moon et al. (2011) found that daytime behaviour had improved following a manualized multi-component behavioural sleep treatment for two out of three children with ASD. CBCL total problem scores were reduced from a borderline range of severity from baseline to an average range at the end of the sleep treatment. These changes were maintained 12-week post-intervention. While this change was identified, it is noteworthy that daytime behaviour appeared to improve following treatment despite no increase in children's sleep duration or improvement in sleep efficiency. It is possible that appropriate bedtime strategies might help to account for this effect. Similar findings were reported by Reed et al. (2009), who found significant improvements in hyperactivity, restricted and self-stimulatory behaviour following sleep treatment.

Most studies suggest that there is some correlation between sleep and challenging behaviour. This relationship could either be a casual or contributory factor (e.g., sleep disturbance causes challenging daytime behaviour) or it may be bidirectional (e.g., sleep disturbance may result in more challenging behaviour or challenging behaviour may result in more sleep problems). More research is required in order to better understand the nature, extent and directionality of this relationship (Cohen, Conduit, et al., 2014; DeVincent et al., 2007; Goldman et al., 2011; Goodlin-Jones, Tang, et al., 2009; Henderson et al., 2011; Hollway & Aman, 2011; Malow et al., 2006; Park et al., 2012; Rzepecka, McKenzie, McClure, & Murphy, 2011; Sikora et al., 2012).

Self-injurious behaviour. SIB's are behaviours that an individual engages in that cause physical harm to his/herself, including self-biting, self-cutting, head banging, self-scratching and many others (Minshawi et al., 2014). Five studies were identified that examined the association between self-injurious behaviour (SIB) and poor sleep in children with ASD (Clements, Wing, & Dunn, 1986; DeLeon, Fisher, & Marhefka, 2004; Goldman et al., 2011; Horner, Day, & Day, 1997; Soke et al., 2017).

Goldman et al. (2011) examined a large cohort of children with ASD for the relationship between sleep problems and self-injurious behaviour (Goldman et al., 2011). They found that of all the behaviours measured on the PCQ, the association between SIB and sleep problems was the strongest. Almost a quarter of poor sleepers had the highest overall risk of exhibiting SIB.

Soke et al. (2017) explored potential associations with SIB in two large samples of children aged between 2-18 years with ASD. This included children from the Autism Speaks-Autism Treatment Network (AS-ATN) which included 4165 participants and the Autism and Developmental Disabilities Monitoring Network (ADDM) which included 8065 participants. The presence of SIB was determined from a parent questionnaire in AS-ATN and from children's records in the ADDM. Sleep was measured using a modified version of the PCQ. They found that SIB was significantly associated with sleep problems.

Two studies have investigated the effect of reducing sleep problems on the frequency of SIB in children with ASD (DeLeon et al., 2004; Horner et al., 1997)

For example, DeLeon et al. (2004) investigated a 4-year-old boy, diagnosed with ASD and developmental delay who displayed SIB primarily within 1 hour of waking. Experimental manipulations which involved manipulating the child's sleep patterns via scheduled awakenings showed that awakenings were functionally related to SIB. In this study, adjustments to the child's sleep patterns through a faded bedtime procedure resulted in an 81% reduction in the frequency of night wakings, and as a result, SIB decreased by 82% (DeLeon et al., 2004). An important limitation of this study is that inter-observer agreement data was only collected during daytime hours and not during the periods in which SIB was likely to occur.

Furthermore, Horner et al. (1997) found that manipulation of sleep schedules using a faded bedtime routine or an inclusion of a daytime nap after a night of reduced sleep due to high rates of SIB reduced the number of nighttime awakenings and associated SIB in a 14-year-old boy. Overall, the mechanism explaining the relationship between SIB and sleep problems are complex, and not yet fully understood (Symons, Davis, & Thompson, 2000).

Adaptive functioning. Adaptive functioning includes the skills needed to complete daily living tasks, motor skills and social skills (Sikora et al., 2012). Knowledge regarding the influence of sleep problems on adaptive functioning in the ASD population is inconsistent and limited. Four studies were identified that have looked at the association between sleep and adaptive functioning in the ASD population (Krakowiak et al., 2008; Richdale & Prior, 1995; Sikora et al., 2012; Taylor et al., 2012).

Taylor et al. (2012) evaluated children with ASD and Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS) from one to ten years of age for the relationship between sleep (measured by the Behavioural Evaluation of Disorders of Sleep), cognitive skills and adaptive functioning (measured by the Vineland Adaptive Behaviour Scale (VABS)). They found that children who slept fewer hours had overall lower adaptive functioning. Specifically, they found that children who slept less on average per night displayed more deficits in skills needed to complete daily living tasks (e.g., pouring water, eating, brushing hair, hygiene, toileting, etc.).

Furthermore, Sikora et al. (2012) found that children with moderate to severe sleep problems had lower VABS composite scores (i.e., lower adaptive functioning) when compared to children with ASD who did not have sleep problems (Sikora et al., 2012). Of note is that sleep problems were related to daily living skills only when sleep problems became moderate to severe. No differences were found between the good sleepers and those

with a mild sleep problem with daily living skills for both pre-school and school-aged children. On the other hand, sleep problems were related to poor socialisation and communication skills regardless of the degree of sleep problems (Sikora et al., 2012).

In contrast to these studies, Krakowiak et al. (2008) and Richdale and Prior (1995) found no association between adaptive functioning, sleep disturbances or sleep duration in children with ASD. For example, Krakowiak et al. (2008) compared parent-reported sleep characteristics in children with ASD between two and five years of age to TD children of the same age as well as children with other DD using a population-based study sample. The VABS was used to determine adaptive and cognitive status. Analyses of the ASD group alone was performed to determine whether total amount of sleep and sleep factors were associated with adaptive functioning. It was found that cognitive level and adaptive functioning did not predict the duration of sleep or the severity of sleep problems (e.g., bedtime resistance, trouble with sleep onset latency).

ASD symptomatology. Many cross-sectional studies of parents of children with ASD have found associations between ASD symptom severity and sleep problems (Allik et al., 2006; Gabriels, Cuccaro, Hill, Ivers, & Goldson, 2005; Goldman et al., 2009; Hoffman et al., 2005; May et al., 2015; Mayes & Calhoun, 2009; Patzold et al., 1998; Tani et al., 2003). However, research findings overall appear inconsistent (Mayes & Calhoun, 2009). Preliminary evidence suggests that sleep problems (e.g., fewer hours of sleep per night) may predict ASD severity scores including more restrictive and stereotypic behaviour (Goldman et al., 2011; Schreck et al., 2004; Tudor et al., 2012). Whereas others have reported a non-significant relationship (Patzold et al., 1998).

Hoffman et al. (2005) investigated the relationship between children's sleep difficulties and the diagnostic criteria for ASD. This study involved 80 children ranging from

4 to 15 years old. Measures used were the CSHQ and the GARS to assess sleep habits, behaviourally based sleep problems and evaluate the degree and probability of ASD. Significant correlations between children's sleep problems and the diagnostic domains of ASD were found. He identified that specific sleep problems reported by parents on their children's sleep problems were related to scores obtained on the GARS. Specifically, sleep-disordered breathing was shown to be the best predictor of children's social interaction problems, stereotyped behaviour and overall level of ASD. Children's parasomnias were another predictor of the GARS domain score, which was the primary predictor of developmental disturbances.

Schreck et al. (2004) evaluated a database of parent-reported sleep problems in 55 children with ASD ranging from 5 to 12 years of age. They found that parental reports of fewer hours of child sleep per night were found to be the primary predictor of increased ASD symptomatology for children. More specifically, those children with increased sensitivity to stimuli in the sleeping environment tended to wake frequently and showed more markedly autistic-type communication patterns on the GARS. Furthermore, children who reacted to their sleeping environments such as light and noise were also more likely to have problems in ASD-related development (e.g., delays in speech development, rocking, regression in skills, etc.). Finally, fewer hours slept per night also predicted difficulties with social interactions (GARS social skills) and overall diagnostic characteristics of ASD (GARS Overall Autism Quotient).

Hundley et al. (2016) explored the association of two types of restricted and repetitive behaviours (RRB), repetitive sensory-motor (RSM) behaviours and insistence on sameness (IS) with sleep problems in children with ASD. Participants included 532 children aged between 2 and 17 years old who participated in the Autism Speaks Autism Treatment Network research registry (Hundley et al., 2016). RSM behaviours were positively associated

with parent-reported sleep problems. This finding remained significant even after controlling for anxiety which is a known predictor of sleep disturbance (Hundley et al., 2016).

Furthermore, Tudor et al. (2012) explored the relationship between sleep problems and ASD symptomatology in a sample of children with a diagnosis of ASD but without any co-morbid diagnoses (e.g., ID, epilepsy and ADHD). Controlling for comorbid conditions was expected to derive a more accurate assessment of the relationship between sleep problems and symptom severity in ASD (Tudor et al., 2012). They found that overall sleep disturbance, short sleep duration and problems with sleep onset delay were associated with all ASD symptoms as well as overall ASD severity.

To better understand the connection between sleep duration and ASD severity, Veatch et al. (2017), analysed ASD-related symptoms using the Autism Diagnostic Observation Schedule (ADOS), the Autism Diagnostic Interview-Revised (ADI-R), Intelligence Quotient (IQ) scores and parent reports of sleep. This data was available in the medical histories of 2,714 children with ASD in the Simmons Simplex Collection (SSC). The SSC represents the largest cohort of autism simplex (where only one person in a family has ASD) families currently collected (Fischbach & Lord, 2010).

ANCOVAS were used, while adjusting for age, to compare ADOS, ADI-R and CBCL scores between children with short versus long sleep duration. Across the SSC dataset, Veatch et al. (2017) observed that increased severity of core ASD symptoms were correlated with decreased sleep duration. Furthermore, more severe RRB and social impairment from the ADI-R were associated with shorter sleep duration when correcting for IQ and age (Veatch et al., 2017).

The most specific and strongest symptoms associated with shorter sleep were a failure to develop peer relationships, and an adherence to non-functional rituals and routines.

Specifically, for every four-minute decrease in sleep duration, there was a one unit increase in the failure to develop peer relationships subscale (Veatch et al., 2017). In addition, shorter sleep duration was associated with more maladaptive behaviours measured via the CBCL and increased reports of obsessive-compulsive disorder, ADHD and depressive disorder. Additionally, when comparing subgroups of children with extremely short and extremely long sleep duration, it was found that children with extremely short sleep duration (in the lower fifth percentile) had increased severity of ASD core symptoms (e.g., RBB and social/communication impairment, lower IQ) and more severe behaviour problems (e.g., anxiety problems, ADHD, anxious/depressed symptoms, sleep problems, problems at school and internalising problems).

A few longitudinal, cross-sectional studies have also found associations between sleep problems, daytime behaviour and ASD symptomatology (Allik et al., 2008; Giannotti et al., 2008; May et al., 2015; Sivertsen et al., 2012). For example, May et al. (2015) measured sleep disturbance at baseline and one year later, they examined change over time and associated problem behaviours. Participants were 84 gender-matched children, aged between 7-12 years old (38 were TD and 46 were diagnosed with ASD. ASD symptoms were reported on the Social Responsiveness Scale (Constantino, 2002), sleep disturbance measured on the CSHQ (Owens, Spirito, & McGuinn, 2000), anxiety measured on The Spence Children's Anxiety Scale (SCAS) (Spence, 1998) and externalising problems on The Conner's Third Edition (Conners, 2003).

This study revealed several key findings. Firstly, there was a decrease in overall sleep disturbance in the ASD group after one year, whereas sleep difficulties in the TD group remained stable (May et al., 2015). Secondly, sleep disturbance predicted later anxiety; this suggests that sleep disturbance may be a risk factor for later anxiety difficulties in both ASD and TD children. Lastly, when the developmental change over one year was examined using

change scores, reductions in ASD symptoms were significantly associated with improved sleep functioning. This association was more pronounced in children with ASD than TD children (May et al., 2015). For the ASD group, a reduction in sleep problems was both associated with an improvement in ASD symptoms and social ability. Only high functioning children were used in this study sample, hence the findings may not extend to children with ID. Overall, this study has extended knowledge on the trajectory of sleep over time in high functioning children with ASD and associated behavioural factors.

These findings suggest that sleep problems are associated with a more severe and intensified repertoire of ASD symptoms during the day, a finding that is consistent with results obtained with wider samples of children with other developmental disabilities (Schreck et al., 2004).

No studies have specifically looked at whether successful treatment of sleep resulted in a reduction in ASD symptoms. However, Reed et al. (2009) found that on the PCQ, significant improvements were observed in self-stimulatory behaviour, while compulsive behaviour showed a non-significant trend toward improvement and on the Repetitive Behaviour Scale, the restricted behaviour scale significantly improved, following a parent behavioural sleep education workshop.

Anxiety. The relationship between sleep problems and anxiety in children and young people with ASD has only recently begun to be explored (Mutluer, Karakoc Demirkaya, & Abali, 2016). Three studies were found that examined this relationship in children with ASD (May et al., 2015; Mazurek & Petroski, 2015; Rzepecka et al., 2011).

Rzepecka et al. (2011) aimed to assess the interrelationship between sleep problems, anxiety and challenging behaviour in the child and adolescent ID/ASD population. This study included 187 parents of children aged 5-18 years of age with ID and/or ASD. They used the

CSHQ, the Spence Children's Anxiety Scale-Parental Version (SCAS-P) and the Aberrant Behaviour Checklist-Community (ABC-C) measures to assess sleep problems, anxiety and challenging behaviour. They demonstrated a significant positive correlation between sleep problems and anxiety, indicating that higher levels of sleep problems are associated with higher levels of anxiety in children with ASD. This is consistent with research with TD children (Alfano, 2007).

In a similar study, Mazurek and Petroski (2015) examined the relationship between sleep problems, anxiety and sensory over-responsivity. Their sample included 1,357 children aged 12 to 18 years old with ASD. The primary measures included the CSHQ, CBCL and the Short Sensory Profile. The results of bivariate analyses indicated that anxiety was significantly associated with sleep problems and there was a positive correlation between anxiety and sleep onset delay, bedtime resistance, sleep anxiety, sleep duration and night-time awakenings (Mazurek & Petroski, 2015). Similarly, sensory over-responsivity was significantly correlated with all sleep problems (Mazurek & Petroski, 2015). No studies have specifically looked at whether reducing sleep problems has resulted in improvement in anxiety in children with ASD.

Cognitive functioning and academic achievement. Over the past decade, sleep researchers have started to decipher the effects of sleep disturbance on daytime cognition in TD populations. For example, a recent meta-analysis by Lim and Dinges (2010) investigated the consequences of sleep deprivation on several cognitive domains and noted performance deficits in attention, working memory and moderate deficits in complex attention (Lim & Dinges, 2010). At present, it is not clear whether sleep disturbance similarly affects these domains in children with ASD (Astill, Van der Heijden, Van Ijzendoorn, & Van Someren, 2012). However, preliminary research has noted that poor sleep quality for children with ASD has correlated with non-verbal intellectual deficits (e.g., non-verbal attention, cognitive

interference) (Elia et al., 2000; Gabriels, Cuccaro, Hill, Ivers, & Goldson, 2005; Roid & Miller, 1997; Taylor et al., 2012), communication problems (Schreck et al., 2004) and academic performance difficulties (Paavonen et al., 2003).

Taylor et al. (2012) found that children with ASD who slept fewer hours on average per a night were more likely to perform worse overall on intelligence and verbal skills than children who slept longer. Moreover, children who slept for fewer hours and suffered from night-time breathing problems showed less ability to complete nonverbal tasks (e.g., puzzles and block building). Gruber et al.'s (2010) observations in TD children also support these results. Taylor et al. (2012) findings are in direct contrast to previous work indicating no relationship between sleep problems and intellectual functioning in children with ASD (Autism Treatment Network, 2010; Diomedi et al., 1999; Krakowiak et al., 2008; Mayes & Calhoun, 2009; Patzold et al., 1998; Wiggs & Stores, 2004).

Moreover, Mayes and Calhoun (2009) examined the relationship between sleep disturbance and comorbid psychiatric symptoms, neuropsychological functioning and academic achievement in 477 children with ASD aged 1 to 15 years old. They found non-significant correlations between sleep problems and math, reading, written expression achievement test scores and neuropsychological test scores (e.g., executive function, working memory, response inhibition, and processing speed) including measures of attention. Also, a non-significant relationship has been found with TD children (Calhoun et al., 2009; Mayes, Calhoun, Bixler, & Vgontzas, 2008). One study found no relationship between neuropsychological functioning (intelligence, attention, executive functioning, processing speed, memory, visual-motor skills, verbal and non-verbal reasoning ability) and sleep-disordered breathing (Calhoun et al., 2009). Similarly, Mayes et al. (2008) found no association between parent-reported sleep problems, overnight polysomnography and academic achievement.

Knowledge regarding the influence of sleep problems on cognition has remained limited due to the focus on night-time disturbances rather than daytime implications. Although the existing research has suggested that sleep problems in ASD may be related to children's cognitive performance, significantly more research within this population must be conducted to understand the impact of sleep disturbance on cognition (Baker, 2011; Taylor et al., 2012). Successful sleep treatments will provide the best knowledge about a causal relation between sleep disruption and impaired performances (Taylor et al. 2012). Overall, findings are inconsistent in the literature regarding the association between cognitive functioning (e.g., IQ), academic achievement and sleep difficulties.

Physical health. Many children with ASD suffer from concurrent medical problems including Gastro-intestinal (GI) problems, obesity, seizures, upper respiratory problems, poor growth, poor appetite, vision problems, hormonal dysfunction, and metabolic disorders (Bauman, 2010; Williams et al., 2004; Yang et al., 2018). Associations have been found between some of these medical symptoms and sleep problems in children with ASD. However, findings are limited and inconsistent (Horvath, Papadimitriou, Rabszty, Drachenberg, & Tildon, 1999; Mannion & Leader, 2013; Mannion et al., 2013b; Williams, Fuchs, Furuta, Marcon, & Coury, 2010; Williams et al., 2004; Zuckerman, Hill, Guion, Voltolina, & Fombonne, 2014). While some research studies report that GI symptoms predict sleep problems (Horvath et al., 1999; Mannion et al., 2013) others studies suggest that sleep problems predicted GI problems (Mannion et al., 2013).

Mannion et al. (2013) investigated predictors of sleep problems in a sample of children and adolescents aged 3 to 16 years old with ASD. They found that the total number of GI symptoms predicted sleep problems. Specifically, abdominal pain predicted sleep anxiety. Avoidant behaviour, undereating, and the 5 GI symptoms (diarrhoea, constipation, nausea, bloating and abdominal pain) predicted daytime sleepiness and parasomnias

(Mannion et al., 2013). Mannion and Leader (2013) found that daytime sleepiness and sleep-disordered breathing predicted both bloating and abdominal pain. In addition, it was found that 67.8% of individuals with ASD had both sleep problems and GI symptoms, while only 8% had neither GI symptoms or sleep problems.

Similarly, Williams et al. (2010) examined GI symptoms lasting more than three months and found that sleep problems occurred more frequently in children with GI symptoms (50%) than those without (37%). Additionally, Horvath et al. (1999) commented that unrecognised GI symptoms may contribute to the behavioural problems of non-verbal children with ASD, such as nighttime awakenings (Horvath et al., 1999).

Furthermore, Williams et al. (2004) found that respiratory problems, vision problems, and a runny nose were associated with decreased sleep time. Vision problems, poor growth and poor appetite were associated with nighttime waking and both poor growth and poor appetite were associated with a decreased willingness to fall asleep.

Lastly, Zuckerman, Hill, Guion, Voltolina, and Fombonne (2014) found associations between poor sleep quality and weight status among children with ASD. Eighty-six percent of obese children, 84% of overweight children and 76% of healthy weight children had clinically significant sleep problems. Obese children with ASD were more likely to have daytime sleepiness, sleep-disordered breathing and total sleep disturbances when compared with healthy weight children. Having obesity is an additional risk factor that makes a child's risk for sleep disturbance extremely high (Zuckerman et al., 2014).

In conclusion, the correlation of medical problems with sleep difficulties is of interest but has unclear significance (Williams et al., 2004) and many studies are inconsistent regarding the directionality between sleep difficulties and medical problems.

Health-Related Quality of Life (HRQoL). HRQoL is a subjective measure of one's overall level of functioning and well-being (e.g., the physical, psychological and cognitive) (Delahaye et al., 2014). Evidence indicates that sleep problems are associated with poorer HRQoL in several different pediatric populations, However, only one study was identified that has explored this relationship in children with ASD and little is understood about the quality of life in this population.

Delahaye et al. (2014) study included 86 parents of children with ASD between the age of 4 and 12 years. They aimed to assess the HRQoL and sleep health of children and investigated the relationship between HRQoL and overall sleep problems. HRQoL was assessed using the parent-report version of the Pediatric Quality of Life Inventory (PedsQL)(Varni, Seid, & Kurtin, 1998), sleep problems were identified using the CSHQ, the CBCL was used to measure behavioural and emotional problems and ASD severity was assessed using the Autism Composite Score of the Pervasive Developmental Disorder Behaviour Inventory (PDDBI) (Cohen, Schmidt-Lackner, Romanczyk, & Sudhalter, 2003). Delahaye et al. (2014) results supported their hypothesis and show a consistent negative relationship between HRQoL and overall sleep problems among children with ASD. Specifically, sleep anxiety, sleep duration and parasomnias as measured by the CSHQ were significantly associated with HRQoL. Additionally, substantial inverse associations were found between total sleep problems and physical and psychosocial well-being (Delahaye et al., 2014). These findings are consistent with other paediatric populations on sleep and HRQoL (Butbul Aviel et al., 2011; Perfect et al., 2012; Sandella, O'Brien, Shank, & Warschausky, 2011). However, there is no existing literature in the ASD population to compare results.

Summary of child outcomes. These studies demonstrate that children with ASD have an increase in stereotyped symptoms and behaviours, communication deficits, social deficits,

self-injurious behaviour, physical symptoms including obesity, GI problems, seizures, upper respiratory problems, poor growth, poor appetite, vision problems, hormonal dysfunction, metabolic disorders and increased internalising and externalising problems such as anxiety, aggression and control problems as well as impacts on adaptive functioning and HRQoL when sleep is disturbed.

Secondary Effects of Sleep Disturbance in parents of Children with ASD

Parental Outcomes

Well-being and ASD. When a child is diagnosed with ASD, parents can experience a multitude of emotional and psychological responses (Wachtel & Carter, 2008). These responses can include grief, loss, depression, stress, shock and denial (Fleischmann, 2004). While a diagnosis of ASD is necessary for early intervention and access to support, it can still be an isolating and distressing outcome for parents. Parents of children with ASD face challenges that can negatively influence their psychological adjustment and mental health (Hodge, Hoffman, Sweeney, & Riggs, 2013; Montes & Halterman, 2007) and they are at increased risk for depression, anxiety and anger (Bristol, Gallagher, & Holt, 1993; Davis & Carter, 2008; Rezendes & Scarpa, 2011).

These rates of mental health problems are not only higher among parents of children with ASD when compared to parents of TD children, but also when compared to parents of children who have Down syndrome, developmental delay and intellectual disability (Abbeduto et al., 2004; Costa, Steffgen, & Ferring, 2017; Duarte, Bordin, Yazigi, & Mooney, 2005; Dumas, Wolf, Fisman, & Culligan, 1991; Estes et al., 2009; Griffith, Hastings, Nash, & Hill, 2010; Hayes & Watson, 2013; Holroyd & McArthur, 1976; Montes & Halterman, 2007; Olsson, Hwang & Högsölan, 2001; Padden & James, 2017; Pisula, 2007; Sanders &

Morgan, 1997; Totsika, Hastings, Emerson, Lancaster, & Berridge, 2011; Weitlauf, Vehorn, Taylor, & Warren, 2014; Yirmiya & Shaked, 2005).

Well-being is decreased in parents in children with ASD and sleep problems exacerbate this (Hoffman, Sweeney, Gilliam, & Lopez-Wagner, 2006; Meltzer, 2011). However, the nature of the relationship between children's sleep problems and parental psychopathology has not yet been determined. Much of the stress associated with parenting a child with ASD compared to other groups (e.g., TD children, Down syndrome) is thought to result from factors directly related to the child's disability (Konstantareas & Homatidis, 1992). For example, dealing with higher frequencies of problem behaviours, and children displaying lower social competence (Benson, 2006; Domingue, 2000; Griffith, Hastings, Nash, & Hill, 2010). High levels of stress and higher levels of unpredictability in caregiving tasks, and insufficient professional support can also negatively impact parents of children with ASD (Bromley, Hare, Davison, & Emerson, 2004; Konstantareas & Homatidis, 1992; Osborne & Reed, 2008; Pisula, 2007; Seltzer, Greenberg, & Krauss, 1995). These challenges can be further compounded by the fact that some parents of children with ASD display aspects of the ASD phenotype, such as social rigidity and weaker central coherence (Griffith et al., 2010). These difficulties may place parents at risk for psychological problems themselves (Griffith et al., 2010). Research suggests that sleep problems affect not only the child but the whole family unit (Malow & McGrew, 2008). Research has shown that parents' mental health and sleep quality is associated with sleep difficulties in TD children (Lam et al., 2003; Shang, Gau, & Soong, 2006) and children with ASD (Chu & Richdale, 2009; Hoffman et al., 2008; Levin & Scher, 2016; Meltzer, 2011). Sleep problems have been found to contribute to depression, anxiety, sleep quality as well as parental stress, over and above symptom severity of ASD (Hodge et al., 2013; Levin & Scher, 2016). Furthermore, researchers have found that improvement of sleep in both TD children and children with ASD

can lead to significant improvements in parent's psychological well-being (Hauck, Hall, Dhaliwal, Bennett, & Wells, 2012; Hiscock et al., 2008; Mindell, Telofski, et al., 2009; Wiggs & Stores, 2001).

Parental Stress. Parents of children with ASD, who report sleep problems, also report frequent daily stress and more hassles related to child rearing (Chu & Richdale, 2009; Doo & Wing, 2006; Goodlin-Jones, Tang, et al., 2009; Hodge et al., 2013; Honomichl et al., 2002; Meltzer & Mindell, 2007; Quine, 1991; Quine & Pahl, 1985; Richdale, Francis, Gavidia-Payne, & Cotton, 2000; Sloper, Knussen, Turner, & Cunningham, 1991).

For example, Doo and Wing (2006) investigated the prevalence of sleep difficulties in children aged 2 to 8 years with PDD and their relationship to parental stress. Parents of 193 children with PDD completed the Parenting Stress Index-Short Form (PSI-SF), the CSHQ and questions on sleep practice. Doo and Wing (2006) demonstrated that sleep problems in children with PDD was a factor leading to increased parental stress. Specifically, they found that 82.6% of mothers and 77.6% of fathers of children with PDD scored above the cut-off point which indicated a significant level of stress in relation to their child's sleep problems.

Levin and Scher (2016) examined the contribution of sleep problems to parenting stress in children with ASD as compared to TD children. Mothers of 34 children with ASD and 31 TD children completed questionnaires measuring sleep-related cognitions, settling to sleep interactions, and the child's sleep problems (Levin & Scher, 2016). Mothers in the ASD group completed a symptom severity questionnaire. Results indicated that children with ASD had more sleep problems than TD children, and their mothers reported higher levels of stress. Children with ASD differed significantly from TD children in that mothers reported more sleep disturbance, particularly more parasomnias, longer sleep onset delay and greater daytime sleepiness (Levin & Scher, 2016).

In another study, Hoffman et al. (2008) examined children's sleep difficulties and severity of ASD in relation to maternal stress levels. Participants involved 72 Mothers and their children with ASD recruited from a centre based behavioural intervention and parent education programme. Maternal reports of their children's sleep problems were related to mothers' reports of their own stress. Children's sleep problems predicted mothers' parenting domain stress and total stress after controlling for mothers sleep and severity of children's ASD. These findings are consistent with research suggesting that the sleep problems of children with ASD may adversely affect their families and contribute to their parent's stress (Cotton & Richdale, 2006; Patzold et al., 1998).

Children's sleep problems may contribute to stress in mothers of children with ASD, but there may be other contributory factors responsible for maternal stress such as mothers own sleep difficulties and their children's ASD symptoms (Hoffman et al., 2008; Richdale, 2003). To date, the relationship between sleep problems associated with ASD and parental stress has received little empirical attention (Hoffman et al., 2008). However, few studies with other developmental disabilities such as Fragile X syndrome have reported a relationship between children's sleep problems and maternal stress (Honomichl et al., 2002; Richdale, 2003).

Parental depression and anxiety. Recent research suggests that there is a specific association between sleep difficulties in children with ASD and maternal depression (Chu & Richdale, 2009; Meltzer, 2011; Meltzer & Mindell, 2007; Shang et al., 2006; Stoléru, Nottelmann, Belmont, & Ronsaville, 1997).

For example, Chu and Richdale (2009) study consisted of 46 mothers and 50 children with developmental disabilities. The SDQ was used to measure children's behaviour problems, the Parenting Hassles Scale (PHS) (Gavidia-Payne, Matthews, Hudson, Richdale,

& Nankervis, 2003) was used to measure the impact of stressors on mothers, the Depression Anxiety and Stress Scale (DASS-21) (Lovibond & Lovibond, 1995) was used to measure maternal depression and anxiety, the Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) was used to measure maternal sleep quality and a children's sleep survey was used relating to the child's diagnosis, sleep problems and severity of sleep problems (Robinson & Richdale, 2004). A Children's Sleep Score (CSS) was derived from the survey. Chu and Richdale (2009) investigated the impact of children's sleep problems on maternal psychological well-being. It was found that greater sleep problems in children were significantly associated with stress, anxiety and depression in mothers. For mothers of children with ASD, the impact of their child's sleep disruption may exacerbate their ability to cope with taking care of the child, which may overwhelm mothers with anxious and depressive thoughts (Chu & Richdale, 2009).

In a further study, Meltzer (2011) examined parent and child sleep as factors associated with depressive symptoms in parents of children with ASD. Families of children with TD children and families of children with ASDs were included in the study if they had a child aged between 4 and 10 years old. Both objective sleep quantity and subjective sleep quality data were obtained using sleep diaries and actigraphs. Parent depressive symptoms were measured by The Centre for Epidemiological Studies Depression Scale (CES-D), child daytime behaviour was measured by the CBCL and children's sleep problems were measured by the CSHQ. It was found that maternal depressive symptoms were associated with shorter child sleep quantity, increased child sleep disturbances, poorer maternal sleep quality and quantity. For fathers, paternal depressive symptoms were associated with increased child sleep disturbances and poorer paternal sleep quality (Meltzer, 2011).

Interventions designed to improve children's sleep may also improve maternal stress, sleep (Eckerberg, 2004; Reid et al., 1999; Wiggs & Stores, 2001) and mental health in TD

children (Hauck, Hall, Dhaliwal, Bennett, & Wells, 2012; Hiscock et al., 2008; Lam et al., 2003; Mindell, Telofski, et al., 2009) and children with intellectual disabilities (Wiggs & Stores, 2001). However, no studies were found that investigated maternal stress and mental health following sleep treatments for children with ASD.

Wiggs and Stores (2001) investigated the mental states of mothers and fathers following behavioural interventions for children with intellectual disabilities. In this study, parents of 15 children with severe intellectual disabilities, challenging daytime behaviour and severe sleep problems received treatment for the child's sleep problem and were compared to 15 controls who received no treatment. Children's sleep problems were assessed by sleep diaries completed by the parents, objective measures of both children's and mothers sleep were made using actigraphs, parental satisfaction with sleep was rated on a 6-point Likert scale, The Malaise Inventory (Rutter, Tizard, & Whitmore, 1970) was used to assess parental stress, The Epworth Sleepiness Scale (ESS) (Johns, 1991) was used to assess daytime sleepiness, The Internality/Externality Control Scale (Rotter, 1966) was used to measure parents' orientation to internal or external control beliefs and Visual Analogue Scales were used to investigate parents' perceived ability to control any difficult sleep-related behaviour shown by their child. Wiggs and Stores (2001) found that successful behavioural intervention in children with severe intellectual disabilities can have a positive impact on both mothers and fathers. For example, mothers in the treatment group reported increased satisfaction over-time with their child's sleep, their ability to cope with their child's sleep and their own sleep. There was also a decrease in daytime sleepiness in mothers seen in both groups. Mother's perceived control over their child's sleep problems had increased in both groups of mothers. Successful effects of treatment on fathers were less modest. Both groups reported increased satisfaction with their children's sleep and their own sleep, but the former to a lesser degree than reported by mothers. There was no change in stress, daytime sleepiness or control over

their child's sleep problems; however, there was an increase in the externality of locus of control in fathers after treatment.

Overall, any causal relationship between children's sleep problems and parents' psychological wellbeing still remains to be explained and there is a shortage of studies addressing this relationship in the ASD population (Hodge et al., 2013; Shang et al., 2006).

Parental sleep quality. Existing research suggests that children's sleep problems impact parental sleep quality in TD children (Boergers et al., 2007) and children with intellectual disabilities (Robinson & Richdale, 2004; Wiggs & Stores, 2001). However, only one study was found that investigated this relationship in children with ASD (Lopez-Wagner et al., 2008). When a child is having difficulties with sleep, usually the mother will wake and tend to the child at night (Glendinning, 1983; Koegel et al., 1992; Meltzer & Mindell, 2007). This parent is, therefore, more likely to have a more disturbed sleep. Mothers and fathers generally respond differently to their child's sleep problems. Differences between maternal reports and paternal reports of sleep and daytime sleepiness have been found.

Lopez-Wagner et al. (2008) investigated 106 parents of children diagnosed with ASD and 168 parents of TD children between the ages of 4 and 16 years old. They used the PSQI (Carpenter & Andrykowski, 1998) to measure adult's sleep quality, the CSHQ to describe sleep habits and identify sleep problems and the GARS-2 to evaluate the probability and severity of ASD. Parents of children with ASD experienced more sleep problems than did parents of TD children. Furthermore, when children's sleep problems were more severe as in the ASD group, parents reported that their own sleep problems were more adversely affected (Lopez-Wagner et al., 2008). Additionally, the severity of children's ASD did not contribute to the prediction of parents' sleep quality in the ASD group.

Children's problematic behaviours may influence the parent-child interactions related to children's sleep (Lopez-Wagner et al., 2008). For example, parents with children with ASD seem to be warier at night due to children engaging in self-injurious or aggressive behaviour (Lopez-Wagner et al., 2008). It may be that these concerns and other nighttime behaviours may contribute to the high level of sleep problems reported by parents in the ASD group (Lopez-Wagner et al., 2008).

No studies were found that looked at change in parental sleep quality post-treatment in children with ASD.

Parental relationship. Parents of children with ASD tend to report less marital satisfaction than parents of TD children (Benson & Kersh, 2011; Lee, 2009). No studies were found that looked at the relationship between marital satisfaction and sleep disturbances in children with ASD. However, one intervention study addressing this association was found in TD children.

Durand and Mindell (1990) study involved a 14-month old girl and her biological parents. Both parents completed the Dyadic Adjustment Scale to measure marital satisfaction. This was administered during baseline, post-treatment and at 1, 2 and 9-month follow-ups. A modified version of graduated extinction was used for this family as the intervention for night waking and bedtime disturbance. At the time of initial assessment, both parents reported being dissatisfied with certain aspects of their marriage. However, following treatment, with an improvement in the child's sleep, an improvement was observed in both the mothers and fathers satisfaction with their marriage (Durand & Mindell, 1990).

The unaffected sibling. A child with ASD and sleep difficulties is often not reserved in letting family members know that they are having trouble sleeping. This often presents as tantrums, screaming or crying. Parents often have to attend to the child's needs at bedtime

and if they wake during the night. The unaffected sibling is possibly woken during this process; this too impacts on the sibling's sleep. This sibling may also feel left out or jealous that most of the attention is getting diverted to the child with sleep problems.

Two studies were found on children with ASD and how their sleep problems impact their TD siblings. Schwichtenberg et al. (2013) explored behaviour and sleep outcomes of TD preschool-aged siblings of children with ASD. Results showed that siblings of children with ASD and sleep difficulties had increased behavioural difficulties measured by the CBCL when compared to siblings of ASD children without sleep problems (Schwichtenberg et al., 2013).

Furthermore, Chou et al. (2012) compared the sleep schedules and sleep problems among children with ASD, their siblings and TD children as well as other factors related to sleep difficulties. A case-control study was conducted, consisting of 110 children with ASD, 125 unaffected siblings and 110 age and sex-matched TD children. All participants were aged between 4-13 years old. The major finding was that children with ASD and their unaffected sibling had more sleep problems and more fixed sleep schedules (e.g., did not demonstrate variable weekday-weekend difference in bedtime, rise time, and sleep duration) compared to children in non-autistic families (Chou et al., 2012). In addition, their unaffected siblings were found to exhibit more sleep talking, higher risk of early insomnia and nightmares compared to TD children in non-autistic families (Chou et al., 2012). A prior finding by Gau et al. (2010) suggests that unaffected siblings of children with ASD experience more severe symptoms of anxiety. In turn, these higher rates of anxiety may explain the higher rates of parasomnias in siblings without ASD (Gau et al., 2010).

Overall, in trying to determine the impact of sleep difficulties some methodological issues impede precise interpretation and need to be considered. One concern is that studies

have used different methods to assess both daytime functioning and sleep. For example, methods range from quantifiable objective recordings to detailed subjective parent reports. Whilst the differences in methodology can make findings difficult to compare across research studies, this difference can also be valuable in highlighting how child sleep disturbances and associated problems can be defined in different yet useful ways (Wiggs, 2007). Lastly, the studies shown are very similar in terms of the cultural groups they include. As discussed earlier, children's sleep patterns and behaviours are strongly culturally determined (McLaughlin Crabtree et al., 2005; O'Connor & Jenni, 2005) and there is a need for cross-cultural research to see how far western societies knowledge about sleep can be applied to other cultures (Wiggs, 2007).

Taken together, these associated problems may make it difficult for the individual and family to function optimally. Given the complicated nature and extent of sleep problems, researchers and clinicians are challenged in identifying reliable interventions that improve sleep in children with ASD. Continuing to focus on sleep is important because effectively resolving sleep problems may directly improve children's well-being and social interactions, as well as decrease caregivers' stress and reduce the number of doctor's office visits (Cavalieri, 2016).

Limitations of Existing Research

Overall, studies of children with ASD are subject to a number of methodological limitations including small sample sizes with research primarily focused on children with high functioning ASD, combined child and adolescent samples, design quality, choice of outcome measures, variations in study designs, absence of a control groups and non-representative samples (e.g., some families lack the resources to take part in research) (Wiggs & Stores, 2001).

Additionally, studies and past evidence are based on parental report of sleep and lack objective measures of sleep (e.g. an infrared video camera, actigraphs) (Williams et al., 2004). Furthermore, a lack of objective measures restricts generalisability and leads to uncertainty of results (Cohen, Conduit, et al., 2014; Delahaye et al., 2014; Turnbull, Reid, & Morton, 2013; Turner & Johnson, 2013; Williams et al., 2004). Multiple and objective methods of sleep assessment are recommended to increase the accuracy of sleep profiles (Hodge, Parnell, Hoffman, & Sweeney, 2012).

Furthermore, one limitation of the literature is we cannot draw on the associations for the maintenance of treatment effects. This is because very few studies of behavioural treatments for sleep disturbance have included data gathered during follow-up phase. This phase is a fundamental methodological component of a research study that helps to assess and analyse the impact and effectiveness of the research as a whole and individual sleep behaviour in the short and long term. More studies need replication and these studies need to be completed with more methodological rigour, this will increase confidence in implementing interventions for families with sleep problems. The long-term efficiency of behavioural interventions for sleep still needs to be determined.

In addition to the limitations, the research to date has mostly focused on associations between sleep problems and challenging daytime behaviour including stereotyped symptoms and externalising behaviours. We cannot determine whether sleep problems cause changes in these outcomes or whether some other unidentified variables are contributing. Additional research is warranted to understand the directionality between sleep disturbance and child and parent outcomes.

Finally, few studies have examined the effects of improvement in sleep outcomes on children's daytime behaviour and parental well-being. Studies are encouraging, however;

there is an insufficient number of intervention studies examining the effects of improvement in sleep outcomes. More research is needed that looks at the benefits and outcomes of interventions that can produce change for these constructs, which may help reduce many of these challenging behaviours in children with ASD.

Rationale

This review has demonstrated the relationship between sleep problems and children's daytime behaviour (e.g., communication deficits, ASD symptomatology, SIB) and aspects of parental well-being (e.g., maternal mood, stress and marital satisfaction). Specifically, these studies have shown an association between sleep and increased ASD symptomatology, medical problems, externalising and internalising behaviours (DeLeon et al., 2004; Doo & Wing, 2006; Hiscock et al., 2008; Mazurek & Sohl, 2016; Reed et al., 2009; Sikora et al., 2012). However, these studies are mostly cross-sectional. Cross-sectional studies only capture an ASD profile at one specific age presentation and do not investigate the directionality between sleep and these secondary outcomes. Therefore, more research is needed to understand the effects of resolving sleep problems using behaviourally based interventions and the impact this has on challenging behaviours, family functioning and parental well-being.

The impact of behaviourally based sleep interventions on parental stress, family functioning and daytime behaviour has not previously been assessed in a systematic fashion (Reed et al., 2009). Only seven studies were found that have investigated the effects of resolving sleep problems using behaviourally based treatments on secondary outcomes in children with ASD (DeLeon et al., 2004; Horner et al., 1997; Loring, Johnston, Shui, & Malow, 2018; Malow et al., 2014; Malow et al., 2006; Moon et al., 2011; Reed et al., 2009). The limited empirical research in this area is possibly because sleep problems may be under-recognised and undertreated because of a greater emphasis on daytime behavioural issues

(Reynolds & Malow, 2011). There is a strong need for more research in this area to address the effectiveness of many behavioural sleep treatments for children with ASD (Adams et al., 2014; Cohen et al., 2014; Mazurek & Sohl, 2016).

This review has demonstrated a range of associations between sleep difficulties and parent and child outcomes for both typically developing children and children with ASD. Most of these studies show negative correlations between children's sleep and challenging behaviour, cognitive functioning and HRQoL, parents sleep quality, parental well-being. Few studies have investigated the effects of behavioural sleep interventions on children's daytime behaviour and parental well-being. Therefore, this study will add to the literature to ascertain the effects that successful sleep interventions have on both parental well-being and challenging behaviour in the ASD population. Additionally, the current research could encourage families to view behaviourally based sleep interventions as a viable option to reduce their child's sleep problems while improving the family's quality of life.

This study has two research questions:

1. Does a behavioural sleep intervention for children with ASD have any effect on their day-time functioning?
2. Does a behavioural sleep intervention for children have any effect on parental well-being in parents of a child with ASD?

Chapter 2

Methodology

This thesis involved the collation and analysis of pre- and post-treatment psychometric assessment data. This was a subset of the data taken from families with a child who had completed an intervention within a larger New Zealand wide sleep and autism study. This chapter describes the current study within the context of the larger scale sleep and autism study.

The Current Study in the Context of the Larger Study

The focus of the larger study was on sleep interventions with children with ASD and sleep problems. The interventions were individualised and based on Functional Behaviour Assessment (FBA). The focus of the current study was to analyse pre- and post- assessment data to determine whether improvement in children's sleep resulting from a behavioural sleep intervention also led to improvement in children's day-time behaviour and in parental well-being.

The sleep Research Team

The research team was led by two senior academics and comprised of registered intern psychologists, PhD and masters students.

Ethics and Consent

Ethical approval was received from the University of Canterbury Human Ethics Committee (#HEC 2014/15). The data used in the current study was gathered under the ethics approval for the larger study. No additional approvals or consents were needed.

All parents were required to give consent for their child to participate in the study. An initial discussion was held on the phone with each family to ensure parents fully understood what the study entailed. The parents and children were provided with written information sheets prior to written consent being obtained (see Appendix A and C). Children were verbally informed of the study and what it involved, and a child consent form was signed by the parents, on behalf of each child. Children indicated assent in accordance with their developmental level.

Design

A single-case multiple baseline across participants design was used as the research design for the larger scale study and a prospective pretest-posttest group design was used for the current study.

A single-case design was chosen for the larger study because of its idiographic nature, allowing an in-depth examination of the effects of interventions on individuals. Each case in the study was largely unique; each treatment plan was exclusive and tailored to each individual case and the presenting complexities.

Pretest-posttest designs are widely used in behavioural research (Dimitrov & Rumrill, 2003), primarily for the purpose of measuring change resulting from experimental treatment (Dimitrov & Rumrill, 2003). One of the advantages of a pretest-posttest design is the simplicity of implementation compared with a true experimental design.

Data Analysis

In the larger study, data obtained from video footage and sleep diaries in baseline, intervention and follow-up phases were graphed for visual analysis, according to the independent variable in each case study. Visual inspection was the primary means of data analysis as a comparison between study phases. Visual analysis of the graphs included assessment of change in trend, level, variability, latency and consistency of change (Cohen, Feinstein, Masuda, & Vowles, 2014).

In the current study, both total scores and subscale scores of the Child Sleep Habits Questionnaire (CSHQ), Child Behaviour Checklist (CBCL), Gilliam Autism Rating Scale (GARS-3), Relationship Quality Index (RQI), Pittsburgh Sleep Quality Index (PSQI), Depression Anxiety and Stress Scales (DASS-21) and the Treatment Acceptability Rating Form Revised (TARF-R) were checked for scoring accuracy and entered into an Excel spreadsheet. Pearson product-moment correlations were computed to determine the relationship between pre-treatment scores for all outcome measures used in the study as well as whether changes in both parent and child outcomes were correlated with changes in children's sleep difficulties post-treatment. Modified Brinley plots (Blampied, 2017) were used to display individual change over time in order to identify systematic effects of the behavioural interventions. Modified Brinley plots are scatterplots that compare individual scores at time one (pre-treatment) with scores at various times post-treatment. The advantage of this analysis is that large amounts of data can be efficiently presented and clearly understood within one figure (Blampied, 2017).

Participants

Participants were 24 (of 42) participants of the larger study who met criteria for the current study.

Recruitment. Participants for the larger study were recruited through organisations throughout New Zealand that provide services to children with ASD, through the networks of the research team or via self-referral.

Inclusion/exclusion criteria. Children were eligible for inclusion in the larger study if they met each of the following criteria: (a) had features of ASD or (b) had a diagnosis of ASD, verified by a paediatrician, registered psychologist, or another appropriate medical professional; (c) were between 2-18 years of age, and (d) had sleep disturbance that consisted of problematic co-sleeping, delayed sleep onset latency, frequent or prolonged night waking's and/or early awakenings as indicated by parent report. Children were excluded from the study if they had a medical condition that impacted on their ability to follow the study procedures or that contributed to their sleep disturbance.

Inclusion/exclusion criteria for the current study. Participants were selected from the larger study if they had completed the following (a) initial assessment, (b) pre-intervention psychometrics, (c) intervention phase, (d) maintenance phase and (e) completed post-treatment psychometrics.

Participants for the current Study. The participants included 4 girls and 20 boys aged between 3 and 14 years old. All children had either features or a diagnosis of ASD. A summary of participant characteristics is presented in Table 1.

Table 1

Summary of Participant Characteristics

Characteristics	Values
Total number of participants	24
Age range	3-14 years old
Mean age	6 years old
Percentage male	83%
Percentage of formal ASD diagnosis	95.8%
Percentage of participants with more than one diagnosis	25%
Sleep problems:	100%
Night waking	38.9%
Co-sleeping	20.4%
Sleep Onset Delay (SOD)	22.2%
Early morning waking	13%
Sleep interfering behaviour	3.7%
Late morning waking	1.8%
Multiple parent-reported sleep problems	79.2%

Measures

Sleep measures. The following measures were only used in the larger study.

Video recordings. Video recordings were collected for a minimum of 30% of baseline and intervention phases of the study and were collected regularly by the researcher. Video recordings helped to triangulate information from the sleep diaries, allowed a more precise measurement of the child's sleep interfering behaviours and parent-child interactions, and were used to calculate inter-observer agreement data.

Parent-reported sleep diaries. Sleep diaries were used to measure sleep behaviours. Parents recorded sleep diaries each night, during all phases of the study. Diaries recorded: (1) daytime sleep (setting, time asleep and time awake), (2) the time the child was put to bed in the evening, sleep onset latency, the frequency of curtain calls (bids for parental attention after the child was put to bed), child behaviour during curtain calls and the parents response to that behaviour; (3), the frequency and duration of night waking's, child behaviour while awake and the parents response to that behaviour, and (4) the time of morning waking. Sleep diaries were collected once a week to monitor progress. Sleep diaries are commonly used in research investigating sleep difficulties (France & Blampied, 2005). See Appendix X for a copy of the standard sleep diary used.

Psychometrics. Data recorded on the following measures were used for the current study

Sleep outcome measures.

The Children's Sleep Habits Questionnaire (CSHQ; Owens, Spirito, & McGuinn, 2000). The CSHQ is the most widely used parent-report measure of sleep in children with ASD (Hodge, Parnell, Hoffman, & Sweeney, 2012). It was completed by one parent in order to classify the type of sleep problem and to ascertain any changes in the presentation of sleep

problems following treatment, as well as to establish whether we had a sample of sleep-disturbed children. The CSHQ is a 45-item parent report instrument that yields both a total sleep disturbance score and eight subscale scores, reflecting key sleep domains that encompass behavioural and medical sleep disorders in the paediatric population: Sleep Onset Delay, Bedtime Resistance, Sleep Anxiety, Sleep Duration, Parasomnias, Night Wakings, Daytime Sleepiness and Sleep Disordered Breathing (Owens et al., 2000). Parents report the frequency of particular sleep behaviours observed in their child over the past week on a three-point scale: usually (5-7 nights per week), sometimes (2-4 nights per week), or rarely (0-1 night per week). The parents noted whether these sleep behaviours were a problem for the family. The CSHQ abbreviated form (CSHQ Abbreviated; Owens, Spirito, & McGuinn, 2000), was also used to measure changes in total sleep difficulties following intervention. The CSHQ is a 22-item parent report instrument that yields a total sleep disturbance score and eight subscale scores. Higher CSHQ scores indicate more parents reported sleep problems. The abbreviated version of the CSHQ was utilised for six out of 24 participants in the study while the full-scale CSHQ was utilised for 16 out of 24 participants.

The CSHQ has good psychometric properties (Hodge et al., 2012; Hoffman et al., 2006; Malow et al., 2014; Malow, Adkins, et al., 2012). Owens et al. (2000) showed adequate internal consistency for both a community sample ($p = 0.68$) and a clinical sample ($p = 0.78$). The CSHQ demonstrated validity by being able to consistently differentiate the community group from the sleep disordered group, yielding a sensitivity of 0.80 and specificity of 0.72. Test-retest reliability was acceptable (range 0.62 to 0.79) (Owens et al., 2000).

The Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI has been used in both research and clinical populations to evaluate adult sleep quality (Carpenter & Andrykowski, 1998). It is also commonly used to research

the sleep quality of parents of children with ASD (Hodge et al., 2013; Hoffman et al., 2008; Lopez-Wagner et al., 2008; McBean, McBean, & Schlosnagle, 2016; Meltzer, 2008).

The PSQI was administered to assess any changes in the quality of parents' sleep over the course of treatment. The PSQI is a 19-item self-report measure used to assess sleep quality and disturbances over a one month time interval in adult populations. Nineteen individual items are grouped into seven component scores: sleep latency, subjective sleep quality, habitual sleep efficiency, sleep duration, use of sleeping medication, sleep disturbances, and daytime dysfunction. Responses were rated according to frequency, with a score of zero indicating the behaviour had not happened during the past month, one referred to the behaviour occurring less than once a week, two indicated it occurred once or twice per week, and three indicated that it occurred three or more times per week. The seven component scores are then summed to yield a global PSQI score, which has a range of 0 to 21; higher scores indicate more impaired sleep.

Buysse et al. (1989) reported good psychometric properties for the PSQI with a test-retest reliability of 0.85 and an internal reliability of 0.83. Also, Backhaus, Junghanns, Brooks, Riemann, and Hohagen (2002) reported an average Global PSQI score test-retest reliability correlation coefficient of 0.87. Global PSQI scores above five resulted in a sensitivity score of 98.7% and specificity of 84.4% for persons with sleep disturbances versus controls (Backhaus et al., 2002).

Parent-wellbeing measures.

Depression Anxiety Stress Scales (DASS-21; Lovibond, Lovibond, & Psychology Foundation of, 1995). The DASS-21 is a widely used instrument and meets the needs of researchers and clinicians who wish to measure the current state or change in state overtime on the three dimensions of depression, anxiety and stress. The DASS-21 has been extensively

used in research with parents of children with ASD (e.g., Al-Farsi, Al-Farsi, Al Sharbati, & Al-adawi, 2016; Giallo, Wood, Jellett, & Porter, 2013). For the current study, the DASS-21 was administered to both parents during the assessment and maintenance phases, to assess overall wellbeing as well as any changes in levels of parental wellbeing. Consistent with the recommendation of Lovibond and Lovibond (1995), the DASS-21 raw scores were doubled so as to make them comparable with data from the full DASS for analysis.

The DASS-21 is a 21-item retrospective self-report measure comprising three scales: (1) a Depression scale, utilizing items that assess dysphoria, lack of incentive and low self-esteem; (2) an Anxiety scale that measures acute responses of fear as well as subjective and somatic responses of anxiety, and (3) a Stress scale that contains items relating to irritability and nervous tension (Clara, Cox, & Enns, 2001). This tool is designed to measure the severity of the core symptoms of depression, anxiety and stress. The parent reports on the severity of a symptom over the previous week on a four-point scale: *Never* (did not apply to me at all), *Sometimes* (applied to me to some degree, or some of the time), *Often* (applied to me a considerable degree, or a good part of the time), *Almost Always* (applied to me very much, or most of the time). The DASS-21 provides an indication of the severity of psychological distress (Henry & Crawford, 2005). The DASS-21 provides scores for each of the subscales which indicates symptom severity; 'Normal', 'Mild', 'Moderate', 'Severe', or 'Extremely severe' (Lovibond & Lovibond, 1995).

The DASS-21 has good psychometric properties (Henry & Crawford, 2005). Henry and Crawford (2005) found that it has adequate reliability for the subscales ($\alpha = 0.82-.90$). Gloster et al. (2008) results indicated good internal consistency, excellent convergent validity, and good discriminative validity, especially for the depression scale.

The internal consistency of each of the DASS-21 subscales is high, 0.94 for depression, 0.87 for anxiety and 0.91 for stress (Antony, Bieling, Cox, Enns, & Swinson, 1998). The DASS-21 also demonstrates strong convergent validity with other measures of depression (e.g., Beck Depression Inventory; $r = 0.79$), anxiety (e.g., Beck Anxiety Inventory; $r = 0.85$) and stress (e.g., State-Trait Anxiety Inventory-Trait; $r = 0.68$) (Antony et al., 1998; Patrick, Dyck, & Bramston, 2010).

The Relationship Quality Index (RQI; Norton, 1983). The RQI is a widely used index of global marital satisfaction (Sanders, Markie-Dadds & Turner, 2001). For this study, the RQI was completed by both parents during assessment and maintenance phases to track any changes in partner satisfaction over the course of treatment. The RQI is a six-item self-report measure given to couples to assess their perception of their relationship satisfaction and quality. Respondents individually rate on a 7-point Likert scale the extent to which they agree with statements about their relationship 1 - very strongly disagree to 7 - very strongly agree). The scores are summed to yield a global relationship satisfaction rating, with higher scores indicating greater satisfaction. The first five items assess relationship satisfaction, strength and stability. The final item assesses overall happiness of the relationship on a 10-point scale ranging from 1 (unhappy) to 10 (perfectly happy).

Measures of daytime functioning.

Child Behaviour Checklist (1 ½-5 years, 6-18 years) (CBCL; Achenbach & Rescorla, 2000). The CBCL is widely used in schools, medical settings, mental health services, child and family services and training programs (Mazefsky, Anderson, Conner, & Minshew, 2011). Many studies have demonstrated a high rate of reliability between the scales of the CBCL and actual psychological diagnosis (Owens et al., 2000). The CBCL has been used to examine the early identification of preschoolers at risk for ASD and research

examining the link between ASD, challenging daytime behaviours and sleep problems, (Goldman et al., 2009; Moon et al., 2011; Muratori et al., 2011).

For this study, the CBCL was administered by the researcher during baseline and maintenance phases to assess children's daytime behaviour. This took approximately 10 minutes for the parents to complete. The CBCL is a 100-item standardised parent-report measure used to assess the child's behavioural and psychosocial difficulties in pre-schoolers aged 18 months to 5 years. Each item describes a specific behaviour and the parent is asked to rate the frequency of behaviour on a 3-point Likert scale: 0 (not true), 1 (somewhat or sometimes true), 2 (very true or often true). Parents are instructed to rate their child's behaviour as it occurs now or how it has been in the two months. The syndrome scales are combined to form *Internalising problems*, *Externalising problems*, and *Total problem behaviours*. T scores are used to determine whether the child's score is in the normal, borderline or clinical range. The CBCL yields seven empirically based syndrome scales: anxious/depressed, emotionally reactive, somatic complaints, sleep problems, withdrawn, attention problems, and aggressive behaviour and five DSM-5-orientated scales: anxiety, depression, oppositional defiant disorder, attention deficit disorder and ASD.

Gilliam Autism Rating Scale-Third Edition (GARS-3; Gilliam, 2016). The GARS-3 was administered during baseline and maintenance phases to identify ASD and estimate its level of severity. The GARS-3 is a 56-item parent or professional report screening questionnaire, it is used for individuals between the ages of 3 and 22. The GARS-3 reflects the DSM-5 diagnostic criteria (American Psychiatric Association, 2013). The subscales are as follows: Social Interaction (14 items), Restricted/Repetitive behaviours (13 items), Emotional Responses (8 items), Social Communication (nine items), Maladaptive Speech (7 items), and Cognitive Style (7 items). Respondents are asked to complete the Likert-type items pertaining to measurable and observable behaviours related to the person being rated. The items are

each rated on a scale from zero to three; 0 (being not at all like the individual), 1 (being not much like the individual), 2 (being somewhat like the individual), and 3 (being very much like the individual). Raw scores are converted to scaled scores which are then summed to yield an Autism Index Score. Higher scores are associated with more severe autistic behaviour. The greater the Autism Index Score is, the higher the probability the individual being rated has ASD and the more severe the autistic behaviour.

The GARS-3 demonstrated superior internal consistency across various age groups. Average Cronbach's alphas of .94 and .93 were recorded for the Autism Index (Gilliam, 2016) including superior test-retest correlations of .90 for the Autism Index. Interclass Correlation Coefficients (ICC) were used to calculate interrater reliability. Average ICC's were in the acceptable range (0.71- 0.85) for all subscales and were considered to be good for the Autism Index (0.84) (Karren, 2017). The GARS-3 has been reported as a consistent and discriminative tool and both sensitivity and specificity are excellent (Karren, 2017).

Treatment acceptability measures.

The Treatment Acceptability Rating Form-Revised (TARF-R; Reimers, Wacker, Cooper, & Deraad, 1992). For the current study, the TARF-R was administered during the maintenance phase. TARF-R is a 20-item parent-report questionnaire used to measure treatment acceptability (Reimers, Wacker, Cooper, & Deraad, 1992). 17 items pertain to general treatment acceptability by asking parents to rate how effective, appropriate and fair they believed the treatment to be, two items address problem severity, and one item surveys their understanding of the treatment (Carter, 2007). The TARF-R is rated on a 7-point Likert Scale. Total scores are obtained by summing all items. Higher summed scores represent greater levels of acceptability.

The TARF-R has good internal reliability, reported to be 0.92 and clinical utility (Finn & Sladeczek, 2001). The TARF-R appears to be a suitable means of assessing the acceptability of treatments proposed in analogue and naturalistic settings (Finn & Sladeczek, 2001). It has been used in an array of settings, including clinical and evaluating treatment effectiveness in the ASD population for example; (Lee, Anderson, & Moore, 2014; McLay, Carnett, van der Meer, & Lang, 2015; McLay, France, Blampied, Danna, & Hunter, 2017).

Post-treatment interview. A semi-structured interview with each of the parents was conducted post-treatment to enhance the researcher's understanding of their level of satisfaction with the outcomes of treatment.

Procedure/study phases

Data was gathered in the original study during the following phases. A summary of study phases are presented in Table 2.

Clinical interview. This followed the format of a standard intake interview used at the Pukemanu Dovedale Clinic and was conducted with the parents by the intern psychologist or researcher, under the supervision of a registered psychologist. Consent and confidentiality procedures were followed, the history and nature of the sleep problem was established and the appropriateness of intervening was decided with the family. The Sleep Assessment and Treatment Tool (SATT) was used to guide the FBA process during the clinical interview with each family. The SATT helps to gain insight into types of sleep problems children are currently experiencing and identifies parent goals as outcome measures (Jin, Hanley, & Beaulieu, 2013). Specific features of the SATT include (a) sleep problems (e.g., night wakings, early morning wakings, sleep onset delay) with a description of any antecedents and consequences (b) the history of the child's sleep problems; (c) sleep goals; (d) child's sleep

routine and schedule; (e) the child's sleep environment; (f) sleep interfering behaviours and sleep dependencies (e.g., events, items).

Assessment. All psychometrics used in the current study, including parent wellbeing measures, sleep outcome measures, and measures of children's daytime behaviour and functioning were administered during the assessment phase.

Baseline. Parents were randomly assigned a baseline length of either one, two or three weeks. During the baseline phase, parents were asked to collect sleep diary data and video recordings each night. Details from the initial clinical interview and baseline data were used to inform the FBA and to subsequently, develop an intervention plan.

Intervention. Sleep diary data and video recordings continued to be collected during intervention. The larger study focused on sleep interventions for children which were individualised based on the outcome of the FBA. Interventions were selected from the following: sleep hygiene/positive bedtime routines, social stories, stimulus substitution, faded bedtime, parental presence, camping out, graduated extinction, consistent sleep/wake times and the use of Gro Clocks.

Stimulus substitution involves finding an appropriate stimulus substitute that replaces the current sleep interfering behaviour but maintains the same consequence for the child. Many children with ASD exhibit sensory seeking behaviours (e.g., seeking movement, textures, or pressure), understanding the sensory qualities that are being reinforced (e.g., by co-sleeping) enables the selection of appropriate stimulus substitutions which can be used to decrease sleep interfering behaviours (Patel, Carr, Kim, Robles, & Eastridge, 2000) (see chapter 1 for definitions of the interventions).

Overall, the goal of treatment was to decrease sleep interfering behaviours and increase sleep-conducive behaviours. Based on the clinical interview and assessments, the researcher developed a treatment plan in collaboration with the family that was unique to the needs of each child and family. Families were provided with daily support through email, telephone or text to ensure that parents remained motivated and confident to carry out the intervention and to resolve any problems that may have arisen. Treatment continued until a notable improvement in target behaviour was observed and the family was satisfied with the level of progress.

Maintenance. The maintenance phase immediately followed the conclusion of intervention and lasted between four to six weeks. All psychometrics and the TARF-R were re-administered during this phase. Other than this, the researchers had no contact with the families. The maintenance phase allowed both child and parent to sustain the newly acquired behaviours in their everyday lives (Blampied, 2013). A semi-structured interview with each of the parents was also conducted post-treatment to enhance the researchers understanding of their level of satisfaction with the outcomes of treatment.

Follow-up. Short-term follow-up data was collected at four weeks post-intervention and long-term follow-up data was collected at 12 weeks post-intervention. During the follow-up phases parents recorded one week of sleep diaries and video footage. The purpose of the follow-up phase was to measure the maintenance of treatment effects across time.

Table 2

Summary of Study Phases

Stage	Larger study	Current study
Clinical interview	Family given psychometrics, sleep diaries, consent, confidentiality, SATT	
Assessment	CBCL, CSHQ, GARS-3, PSQI, DASS-21, RQI	CBCL, CSHQ, GARS-3, PSQI, DASS-21, RQI
Baseline (between 1-3 weeks)	Sleep diary data, video footage	
Sleep treatment (variable time period)	Sleep diary data, video footage	
Maintenance (4-6 weeks)	Re-administered CBCL, CHSQ, GARS-3, PSQI, DASS-21, TARF-R	Re-administered CBCL, CHSQ, GARS-3, PSQI, DASS-21, TARF-R, RQI
Follow-up (1 week)	Post-treatment interview, sleep diary, video footage	

Results

Chapter 3

This chapter presents data comparing pre- and post-treatment measures of children's daytime functioning and parental well-being for 24 children who received a behavioural sleep treatment and their parents. The chapter will begin by presenting data in tables that compare pre- and post-treatment scores on the CSHQ, GARS-3, CBCL, PSQI, RQI and the DASS-21. Frequency distributions show pre- and post-intervention scores for all child and parent outcomes. Pearson product-moment correlations were computed to determine the relationship between pre-treatment scores for all outcome measures used in the study as well as whether changes in both parent and child outcomes were correlated with changes in children's sleep difficulties post-treatment. Modified Brinley plots (Blampied, 2017) were used to identify systematic effects of treatment. Lastly, treatment acceptability data that was collected post-intervention is also presented in a table.

For 16 out of 24 children, the child's mother and father completed the CSHQ, CBCL, GARS-3, PSQI and the DASS-21. In seven cases, as the mother or grandmother was a solo parent, only maternal data was available and in one case, as the father was the solo parent, only paternal data was available. Additionally, two children were siblings and had the same parents. Some measures did not apply for all participants, for example, the RQI; this was only completed by nine couples.

Quality of Data

The GARS-3 was not administered to 7 out of 24 children with one of these children missing pre-intervention scores. One of the 24 children's parents were missing mothers post-scores on the DASS-21, and six out of 24 children's parents were missing full data sets for the PSQI. Of these six, one participant was missing fathers' scores so mothers' scores only

were reported for this family. Lastly, seven participants were not administered the CBCL.

Any participant who had not completed full sets of psychometrics was omitted from the modified Brinley plot analysis. On the CSHQ modified Brinley plot, 1 out of 24 participants' pre- and post-intervention scores were not plotted due to the administration of two different versions of the CSHQ. Additionally, the CSHQ could not be administered to one participant because of the child's age, which was outside of the validated age range for this measure.

Lastly, Six children were administered the CSHQ abbreviated version.

Child Sleep and CSHQ, GARS-3, and CBCL Measures

Table 3

Means and Standard Deviations for Children's Outcome Measures

Child outcomes	Pre				Post					
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>M</i>	<i>SD</i>	<i>Range</i>	<i>d_{av}</i>	95% CI
CSHQ	16	52.13	7.91	27	16	44.88	6.02	22	-1.03	-1.68, -0.36
CSHQ (Abbrev)	6	30.33	9.83	27	6	17.00	8.56	21	N/A	N/A
GARS-3	16	105.69	8.36	33	16	98.94	14.70	47	-0.56	-1.05, -0.06
CBCL (1 1/2-5 years)	9	58.89	16.93	40	9	58.89	17.77	48	-0.49	-0.79, -0.19 (CBCL pooled data – Externalising behaviours)
CBCL (6-18 years)	8	72.00	3.91	12	8	66.67	6.58	16	-0.17	-0.42, 0.09 (CBCL pooled data – Total behaviour)

Note. N= Participants, M = Mean, SD = Standard Deviation, *d_{av}* = Cohen's *d* Effect size, 95% CI = 95% Confidence Interval, N/A = Not Assessed

Table 3 displays the means, standard deviations and Cohen's d_{av} Effect Size for pre – and post-treatment scores on the CSHQ, CSHQ abbreviated form, GARS-3, and the CBCL for each of the 24 participants.

Prior to treatment, the average score on the CSHQ (52) was within the clinical range. Following treatment, the average score reduced to an average of 45, still in the clinical range.

The average score on the abbreviated CSHQ (30) reduced pre to post-treatment to (17), but the lack of reference data for this measure prevents interpreting this with reference to clinical a cut-off.

Average GARS-3 scores (105) pre-treatment fell within the level 3 ASD severity range requiring “very substantial support”. Following treatment, this reduced to an average of 99, a reduction to Level 2 ASD severity range, requiring “substantial support”.

Prior to treatment, the average score on the CBCL (1 ½ - 5 years) (59), was within the “normal” range and this did not change following treatment. Average pre-treatment score on the CBCL (6 – 18 years) (72), fell within the clinical range, and while the average score fell (67), it remained within the clinical range post-treatment.

Parent PSQI, RQI and DASS-21 Outcome Measures

Table 4

Means and Standard Deviations for Parent Outcome Measures

Parent outcomes	Pre				Post					
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>M</i>	<i>SD</i>	<i>Range</i>	<i>d_{av}</i>	<i>95% CI</i>
PSQI	25	8.60	3.71	15	25	7.64	3.98	15	-0.25	-0.61, 0.11
RQI	20	36.35	10.12	36	20	36.80	7.51	26	0.05	-0.20, 0.30
DASS-Dep	38	10.03	8.89	36	38	7.50	7.38	30	-0.30	-0.60, -.004
DASS- Anx	38	6.87	6.36	24	38	4.87	5.30	26	-0.34	-0.63, -0.05
DASS- Stress	38	16.34	9.44	37	38	14.05	8.30	38	-0.26	-0.53, 0.02

Table 4 provides the means and standard deviations for pre – and post-treatment scores on the PSQI, RQI and DASS-21 for the parents of each of the 24 participants.

Prior to treatment, the average score on the PSQI (9) was within the clinical range. Following treatment, the average score reduced to an average of 8, this remained within the clinical range.

Prior to treatment, the average score on the RQI was 36. Following treatment, this reduced to an average of 37.

Average DASS-21 Depression subscale scores (10) fell within the “mild” range. Following treatment, this reduced to an average score of eight, a reduction to the ‘normal’ range.

Average Anxiety subscale scores on the DASS-21 (7) fell within the “normal” range. Following treatment, this reduced to an average score of (5) and while the average score fell, it remained within the “normal” range.

Average stress subscale scores on the DASS-21 (16) fell within the “mild” range. Following treatment, the average score reduced to 14 and fell within the “normal” range.

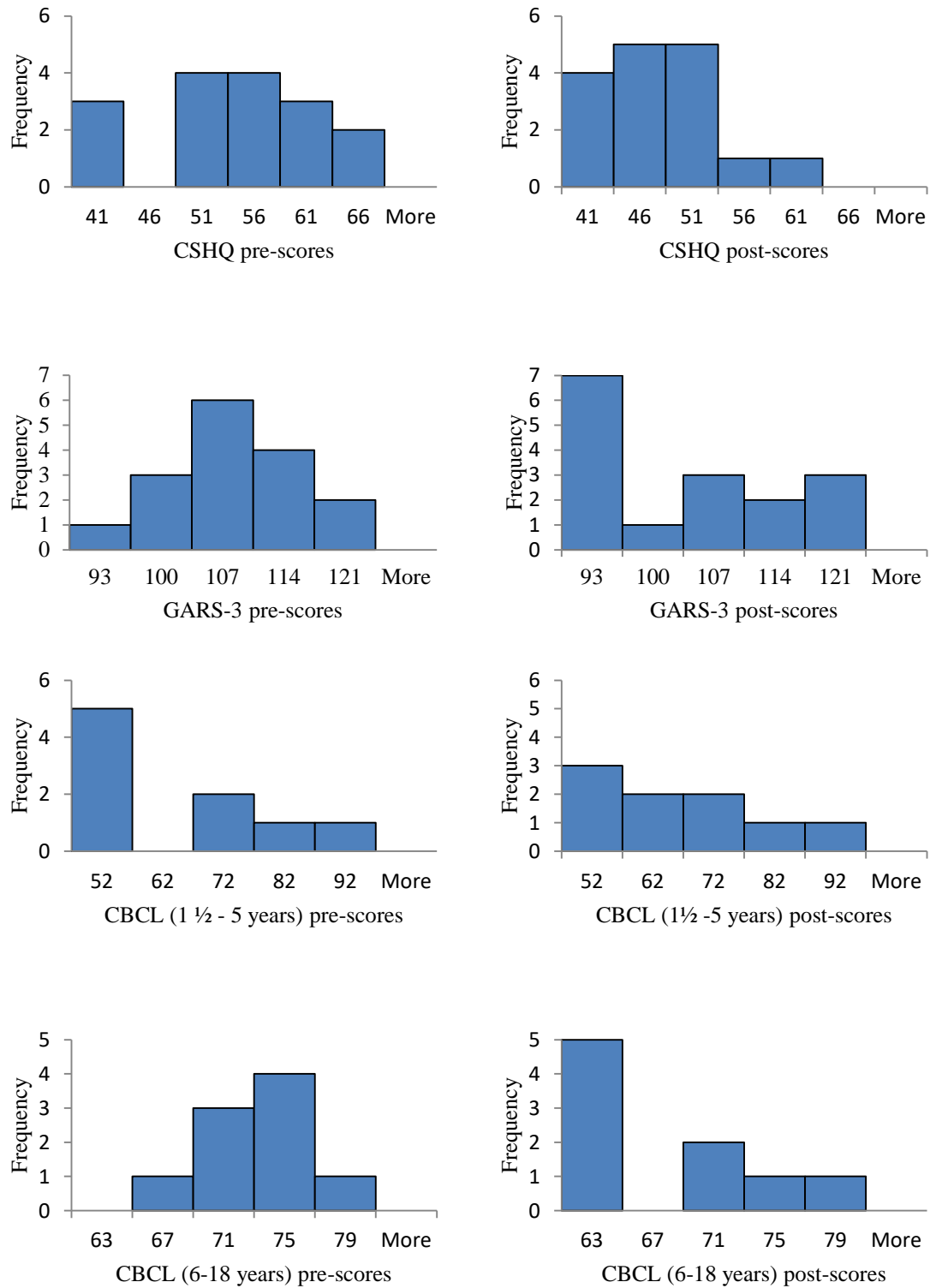


Figure 1. Distribution of Pre- and Post-intervention Child Outcomes scores on the CSHQ, GARS-3 and the CBCL.

Figure 1 displays the frequency distributions for pre and post-intervention scores on all child outcomes. Frequency distributions provide for checks in skew, outliers, multi-modal distribution, and evidence of non-normality to the data. Scores on the CSHQ have shifted from a relatively normal distribution to the majority of scores being distributed near the lower end of the scale (between 41 and 51) indicating a reduction in sleep difficulties. The GARS-3 pre-intervention scores showed a normal distribution with the majority of scores clustered between 101 and 107. Post-intervention shows the majority of scores to be clustered below 93, this falls within the level 2 ASD severity requiring “substantial support”.

The CBCL (1 ½ -5 years) pre-intervention scores shows a cluster of scores toward the lower end of the scale between 43 and 52, this is within the “normal range”. Following intervention, CBCL scores showed little change with the majority of scores remaining between 43 and 52. The CBCL (6-18 years) pre-intervention scores showed a relatively normal distribution with a cluster of scores falling between 72 and 75, falling within the clinical range. Following intervention, the majority of CBCL total scores were positively skewed and shifted toward the lower end of the scale between 60 and 63, falling in the “borderline clinical” range.

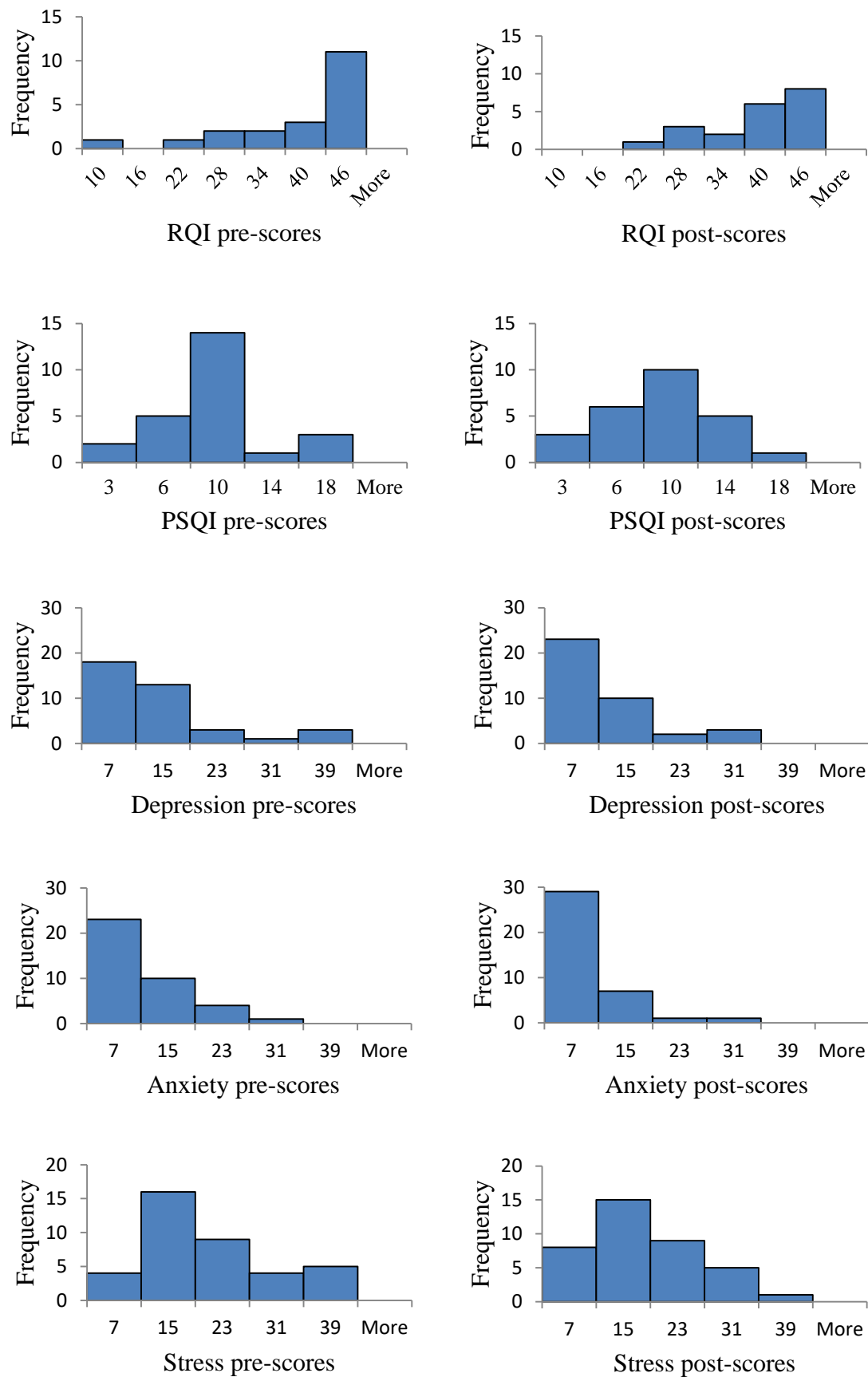


Figure 2. Distribution of pre- and post-intervention parent outcome scores on the RQI, PSQI and the Depression, Anxiety and Stress subscales of the DASS-21.

Figure 2 displays the frequency distributions for pre- and post-intervention scores on all parent outcomes. The RQI showed both pre- and post-intervention scores to be negatively skewed toward higher scores, with pre-intervention and post-intervention scores between 41 and 46 for 9 out of 18 parents. This indicates a high level of overall relationship satisfaction within the study participants both at pre- and post-intervention. One extreme score between 5 and 10 for one participant was observed at pre-intervention, this indicated a lower relationship satisfaction compared to other participants in the study. For the depression and anxiety subscales on the DASS-21, the majority of scores were clustered below seven, reflecting the majority of participants to be within the normal range. Following intervention, depression scores showed a positive skew. Anxiety scores showed little change following intervention. The stress subscale for both pre- and post- intervention were clustered between 8 and 15, this was within the normal to mild range. The PSQI scores showed a normal distribution both pre- and post-intervention with the majority of scores clustered between a global score of 7 and 10.

The Relationship Between the CSHQ, CBCL, GARS-3, DASS-21, PSQI and the RQI

Table 5

Pearson Product Moment Correlation of Pre-Intervention Psychometric Scores

Variable	1	2	3	4	5	6	7	8	9
1. CSHQ Full Scale	1	0.06	-.633*	-0.60	0.44	-0.28	.706**	0.37	-0.05
2. GARS-3		1	0.45	0.59	0.49	-0.23	-0.11	-0.01	-0.58
3. RQI Mother			1	.750*	-0.15	-0.05	-0.43	-.674*	-0.12
4. RQI Father				1	-0.09	0.04	-0.55	-0.40	-0.15
5. PSQI Mother					1	0.49	0.25	.590*	-0.17
6. PSQI Father						1	-0.15	0.24	0.65
7. CBCL (1½-5 years & 6-18 year forms)							1	0.39	-0.24
8. DASS-21 Mother								1	0.07
9. DASS-21 Father									1

Note. Correlations printed in bold are significant; *p < .05., **p < .01.

In order to examine the relationships between outcome measures, Pearson product-moment correlation coefficients were computed to determine the relationship between pre-treatment scores for all child and parent outcome measures used in the study. These correlations are presented in Table 5. Results showed statistically significant correlations (i.e., correlations > 0) between five scales. In five instances correlations were large enough to reject the null hypothesis that true $r = 0$. Poor children's sleep was negatively correlated with mothers' relationship quality and positively correlated with CBCL scores. The parents' relationship quality was moderately positively correlated, as expected. The total DASS-21 scores were moderately negatively correlated with mothers' relationship quality and moderately positively correlated with mothers sleep quality.

A significant positive correlation was found between the CSHQ scores and the CBCL scores indicating increased severity of the sleep problem was correlated with increased severity of the children's problematic daytime behaviour. This correlation was in the direction expected and the effect size for this analysis was found to exceed Cohen's convention for a medium effect size.

A significant negative correlation was found between the CSHQ scores and mothers RQI scores, indicating increased ratings of sleep problem severity were correlated with a decrease in maternal reports of relationship quality. This correlation was in the direction expected and the effect size for this analysis was found to exceed Cohen's convention for a medium effect size.

A significant positive correlation between mothers RQI scores and fathers RQI scores was found, indicating high ratings of maternal relationship satisfaction were correlated with high ratings of paternal relationship satisfaction. This relationship was in the expected

direction and the effect size for this analysis was found to exceed Cohen's convention for a medium effect size.

A significant negative correlation between mothers RQI scores and mothers DASS-21 scores was found, indicating that lower ratings of maternal depression, anxiety and stress levels were correlated with higher maternal ratings of relationship quality. This relationship was in the expected direction and the effect size for this analysis was found to exceed Cohen's convention for a medium effect size.

Lastly, there was a significant positive correlation between mothers' scores on the PSQI and the DASS, indicating increases in mothers' depression, anxiety and stress was correlated with increases in mothers' sleep difficulties. This relationship was in the expected direction and the effect size was found to exceed Cohen's convention for a medium effect size.

No significant correlations were found between the CSHQ and the RQI (fathers scores), PSQI (mothers and fathers scores), DASS-21 (mothers and fathers scores) or between the GARS-3 and the RQI, PSQI, CBCL and the DASS-21.

No significant correlations were found between RQI and the PSQI, CBCL, and the DASS-21 (except for a significant correlation between mothers RQI scores and mothers DASS-21 scores), or between the PSQI and the DASS-21, CBCL and the RQI and lastly no significant correlations were found between the DASS-21, the CBCL and PSQI (fathers score).

Table 6

*Pearson Product Moment Correlation of CSHQ Change Scores and Parent and Child**Outcome Measures*

Variable	CSHQ Change Score	GARS-3	CBCL	RQI M	RQI-F	PSQI- M	PSQI - F	DASS- 21 M	DASS-21 F
CSHQ Change Score	1	-0.29	-0.19	0.46	0.07	-0.29	-0.17	-0.13	0.12

Note: M = Mother F = Father

Pearson product-moment correlation coefficients were computed to determine whether change in children's sleep from pre- to post intervention as measured by the CSHQ was significantly correlated with changes in the DASS-21, RQI, PSQI, CBCL and the GARS-3. The outcomes of this analysis are presented in Table 6. The results revealed a non-significant trend in the predicted direction. Correlations were small to moderate indicating that more change in children's sleep from pre to post –intervention correlates with more change in both child and parent outcome variables at post-treatment.

Modified Brinley Plots Interpretation

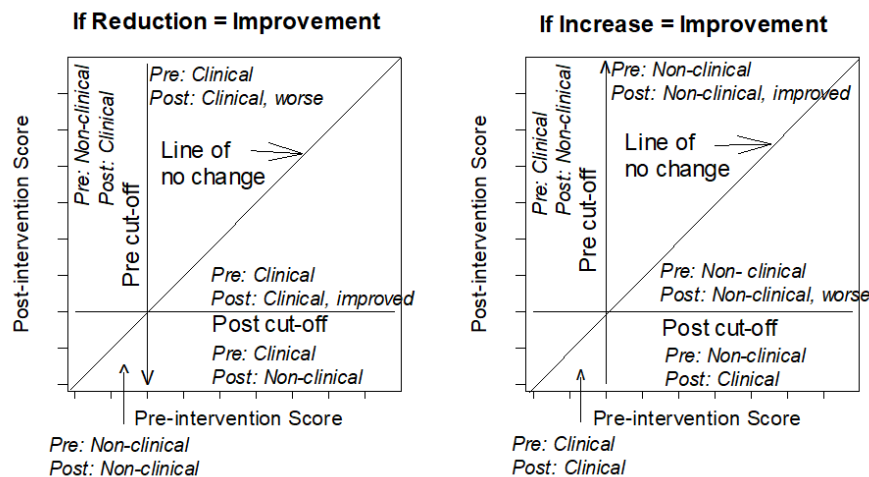


Figure 3: Interpretation of the graph zones in modified Brinley Plots when reduction (left-hand graph) or increase (right-hand graph) in score represents clinical improvement (Blampied, 2017).

As seen in Figure 3, modified Brinley plots are used to display individual change over time in order to identify systematic effects of an intervention. Each individual's data are displayed as a coordinate pair on a scatter plot with pre-intervention scores plotted on the X-axis and post-intervention scores on the Y-axis. Therapeutic change, if any, is revealed by data deviations about the 45-degree diagonal line of no change. Data points will lie on or near the 45-degree diagonal lines of no change ($X = Y$) if there are no systematic differences between pre- and post-intervention scores. If there are systematic differences between pre and post-intervention, the data points will deviate from the line (either above or below) (Gordon, Rucklidge, Blampied, & Johnstone, 2015). When a higher score indicates greater impairment, points that fall above the central diagonal line indicate greater impairment and those below the line indicate less impairment (Figure 3). When a higher score indicates better functioning, the reverse holds true.

Interpretation is assisted by displaying an arrow to indicate the direction of desired change, and by showing clinical cut-offs if they exist for particular measures (Gordon et al.,

2015). As seen in Figure 3, the zones on the graph created by the intersections of the cut-off lines and the diagonal line of no change can be interpreted in clinical terms.

Each participant's degree of change from pre- to post-intervention was classified using the RCI (Reliable Change Index) (Jacobson & Truax, 1991). The RCI is based on the SE_M (Standard Error of the Mean) for each measure, and indicates how much change is required for a change score to lie outside the range expected because of measurement error alone. On the modified Brinley plots, the upper and lower bounds of the RCI are shown as lines parallel to the no-change line. Individuals whose data points lie within these boundaries have not shown reliable change. Further, the phase mean values and 95% CI for the mean maybe displayed as crosses on the graph, with the centre of the cross at the coordinates of the X and Y means and the length of each line of the cross indicating $\pm 95\%$ CI of the relevant mean.

Two Effect Size (ES) measures were computed: Cohen's d_{av} (Lakens, 2013) and the Probability of Superiority (PS) also known as the Common Language Effect Size (McGraw & Wong, 1992). Strictly, Cohen's d is a family of effect sizes and subscripts should be used to indicate the particular member of the family represented (Lakens, 2013). For pre-post, within-subject data, this is d_{av} , since the standardizer is the average of 16 pre and post standard deviation (Gordon et al., 2015; Lakens, 2013). Since only d_{av} is reported in this thesis, the subscript is omitted. Positive values of d reported subsequently indicate change in a clinically desirable direction if increases in scores reflect improvement on a measure. Negative values of d reported subsequently indicate change in a clinically desirable direction if decreases in scores reflect improvement on a measure. Interpretation of effect sizes for this thesis is based on benchmarks suggested by Cohen (1988). Effect sizes are interpreted as small ($d = 0.2$), medium ($d = 0.5$), and large ($d = 0.8$). The PS represents the probability (reported as a percentage) that, for any randomly selected participant, their second (post)

score is clinically better than their first (pre) score (Lakens, 2013; McGraw & Wong, 1992), and was calculated using Lakens (2013) software. Confidence intervals about d (calculated using (Cumming, 2012)) were used to classify d as reliably different from zero ($p < 0.05$).

Modified Brinley Plots – Child Outcomes

The CSHQ, CBCL and GARS-3 scores are presented below as modified Brinley plots showing individual change from pre- to post-intervention.

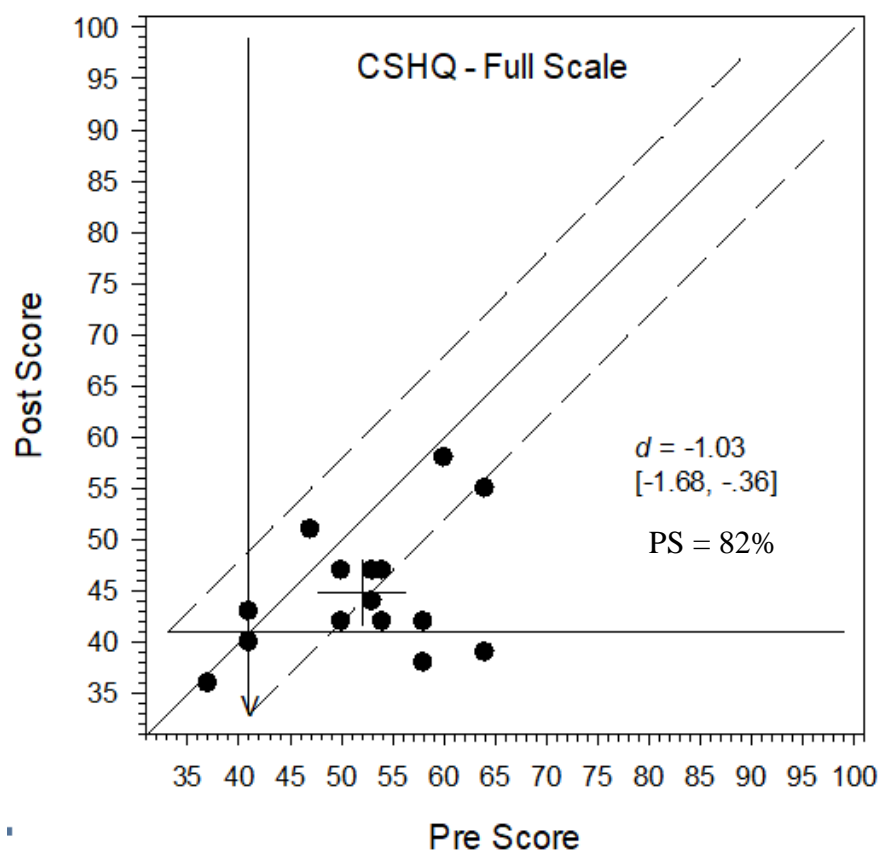


Figure 4. Modified Brinley plot showing change from pre- to post- intervention on the full-scale CSHQ. Both vertical and horizontal lines placed to show the clinical cut-off value. The arrowhead on the vertical cut-off indicates direction of desired change. The cross marks the coordinate point of the means on each measure and the length of the cross arms show the 95 % confidence interval on the respective mean. d = Cohens d effect size; PS = Probability of Superiority.

CSHQ. As seen in Figure 4, 15 out of 16 children had total sleep difficulties within the clinical range (between 41 and 99) pre-intervention. Of these children, two showed an increase in total sleep difficulties while 13 showed a reduction in such difficulties post-treatment. Six out of 16 children showed reliable improvement with three of these cases falling within the non-clinical range suggesting a clinically significant improvement following intervention. One child had sleep difficulties rated in the non-clinical range pre-intervention with negligible change post-intervention. Using Cohen's d , the ES was large for the CSHQ ($d = -1.03$) and reliably different from zero (95% CI [-1.68, -.36]).

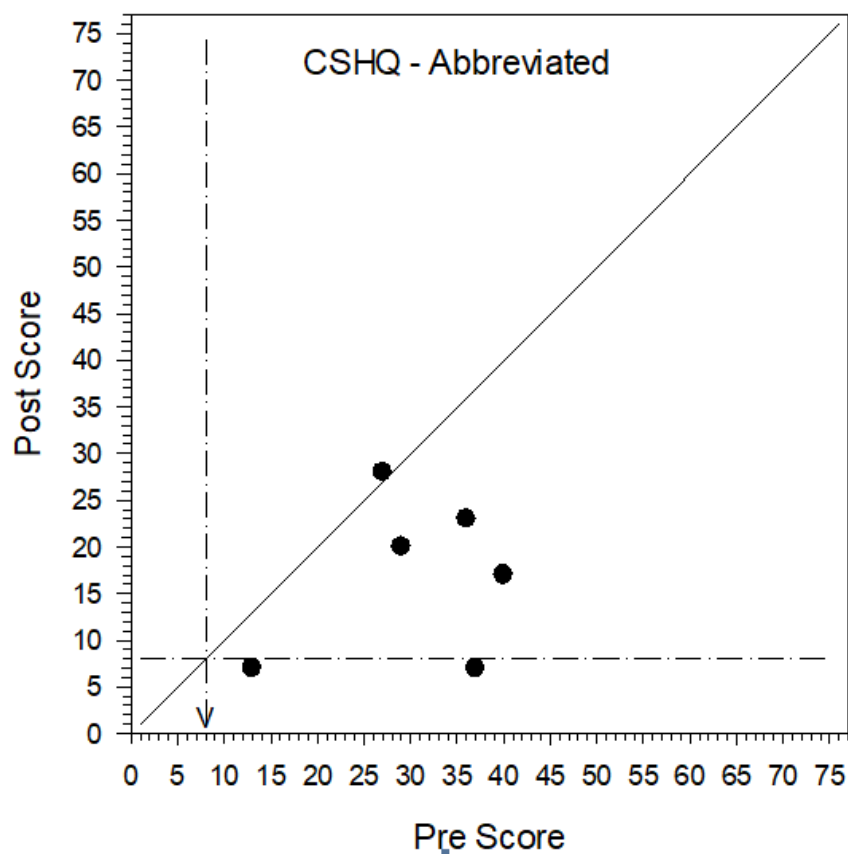


Figure 5. Modified Brinley plot showing change from pre- to post- intervention on the abbreviated version of the CSHQ. Both vertical and horizontal lines are placed to show the clinical cut-off value. The arrowhead on the vertical cut-off indicates the direction of desired change. The cross marks the coordinate point of the means on each measure and the length of the cross arms show the 95 % confidence interval on the respective mean. The clinical cut-off lines are dashed – dot to indicate that this is set at 8 by extrapolation (41-33), not by psychometric analysis.

CSHQ Abbreviated version. As seen in figure 5, all six children pre-intervention fell within the clinical range (above 8). Five of these children showed improvements in total sleep difficulties, and of these children, two fell within the non-clinical range showing a significant improvement. One child showed a small increase in total sleep difficulties following intervention.

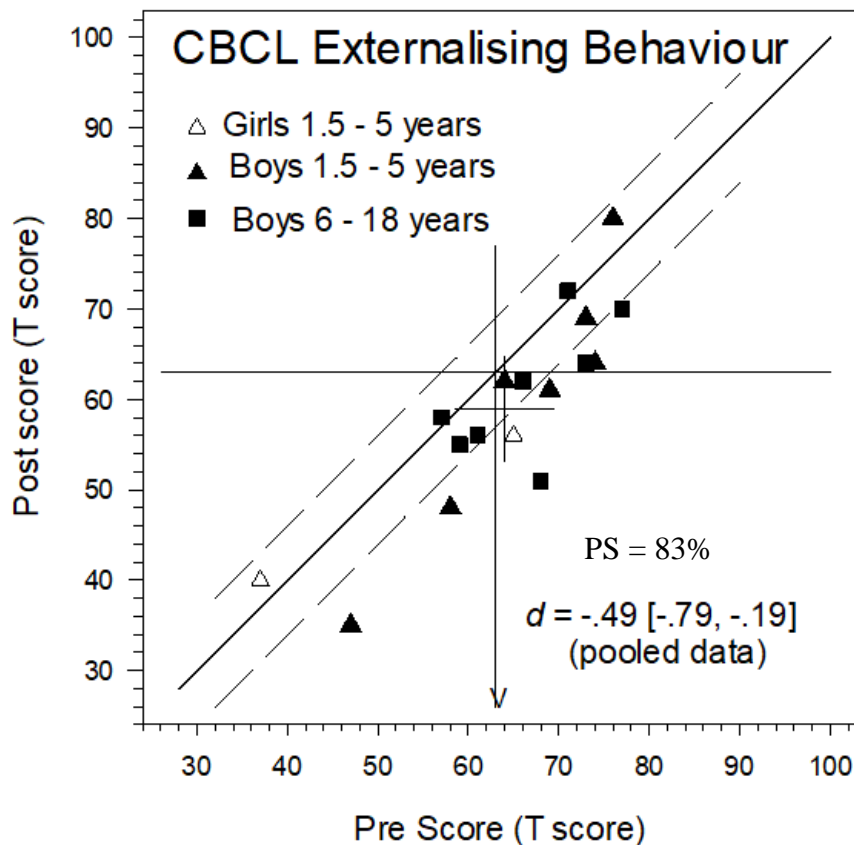


Figure 6. Modified Brinley plot showing change from pre-intervention to post-intervention on Externalising Behaviours on the CBCL outcome measure. Both vertical and horizontal lines placed to show the clinical cut-off value. The arrowhead on the vertical cut-off indicates direction of desired change. The cross marks the coordinate point of the means on each measure and the length of the cross arms show the 95 % confidence interval on the respective mean. d = Cohens d effect size; PS = Probability of Superiority.

CBCL Externalising Behaviours. As seen in Figure 6, 11 out of 17 children scored at pre-intervention in the clinical range (T score > 63) on the externalising behaviours subscale of the CBCL. Of these 11 children, six remained in the clinical range following

intervention, and of these six, four showed a decrease in externalising behaviour; one case showed a slight increase in externalising behaviour and one case remained unchanged following intervention. Five out of the 11 cases moved from the clinical to non-clinical range. Of these five cases, three cases showed improvement in externalising behaviour to a clinically significant degree following intervention.

Six children were in the non-clinical range at pre-intervention and all remained in the non-clinical range post-intervention. Of the children who remained in the non-clinical range at post-intervention, one case showed no change in externalising behaviour, one case showed an increase in externalising behaviour, while two children improved to a clinically significant degree following intervention. The remainder showed little change. The ES was small for the CBCL Externalising behaviour scale ($d = -.49$) and reliably not zero (95% CI [-.79, -.19]).

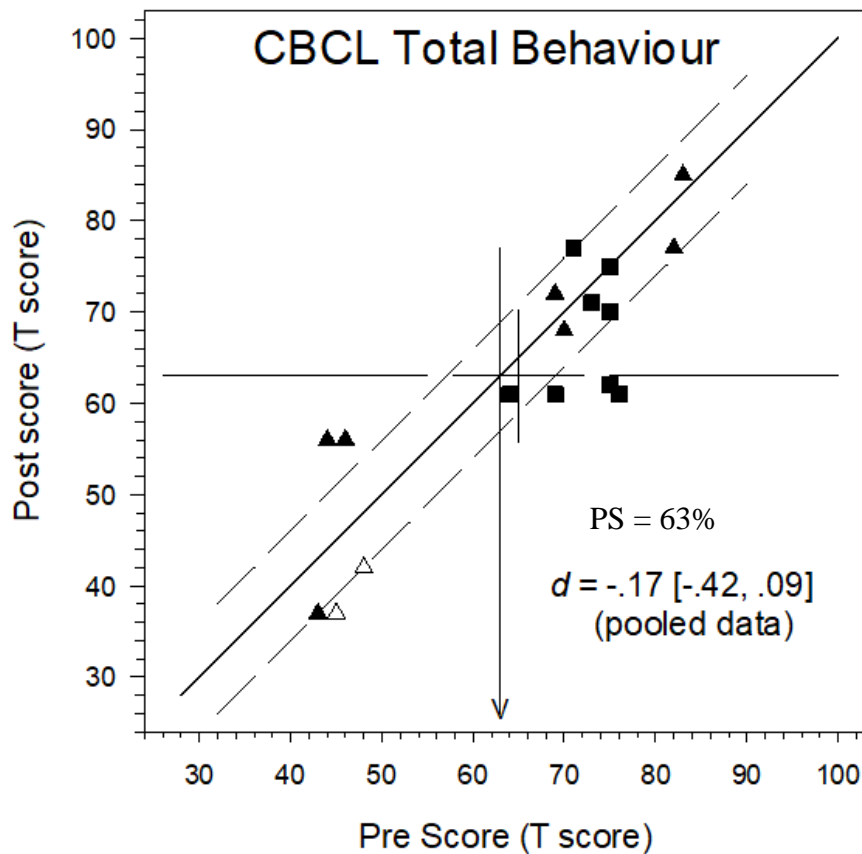


Figure 7. Modified Brinley plot showing change from pre- to post-intervention on Total Problem Behaviours on the CBCL outcome measure. Both vertical and horizontal lines placed to show the clinical cut-off value. The arrowhead on the vertical cut-off indicates direction of desired change. The cross marks the coordinate point of the means on each measure and the length of the cross arms show the 95 % confidence interval on the respective mean. d = Cohens d effect size; PS = Probability of Superiority.

CBCL Total Problem Behaviours. As seen in Figure 7, 12 out of 17 children scored within the clinical range (T score > 63) on Total Problem Behaviours pre-intervention. Of these 12 children, eight remained in the clinical range following intervention. Of these, one child showed no change, three children showed an increase in total problem behaviour and four children slightly improved following intervention. Four of the 12 children in the clinical range pre-intervention moved into the non-clinical range post-intervention and three children improved to a clinically significant degree following intervention.

Five children were in the non-clinical range prior to treatment. This was maintained post-treatment. Of the children who remained in the non-clinical range, two showed deterioration in scores indicating an increase in problem behaviour to a significant degree. Three out of five cases improved and of these three, one showed reliable improvement indicating a decrease in total problem behaviour following intervention. The ES was very small for the CBCL Total Behaviour Problems ($d = -.17$) and it was not statistically significantly different from 0 (95% CI $[-.42, .09]$).

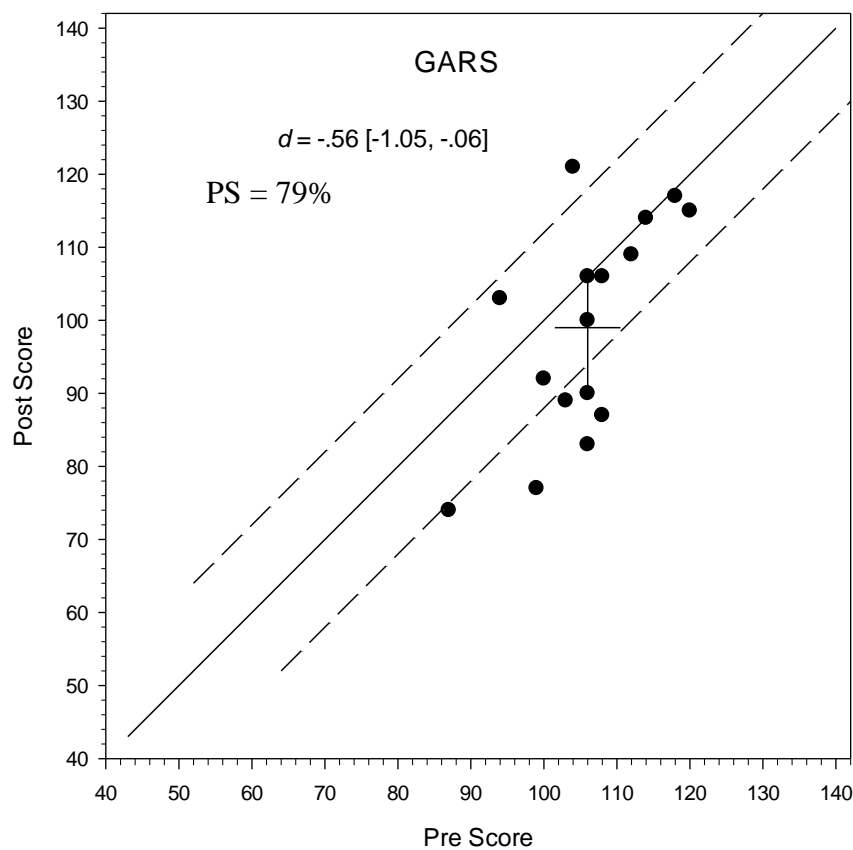


Figure 8. Modified Brinley plot showing change from pre- to post-intervention on the GARS-3 outcome measure, with higher scores indicating a high level of ASD severity. The cross marks the coordinate point of the means on each measure and the length of the cross arms show the 95 % confidence interval on the respective mean. d = Cohens d effect size; PS = Probability of Superiority.

GARS-3. As seen in Figure 8, 13 out of 16 children had an ASD index score within the level 3 range, consistent with requiring very substantial support prior to intervention while 3 of the 16 had an ASD index score within the level 2 range requiring substantial support. Twelve out of 16 participants showed a decrease in ASD Index scores indicating a reduction in ASD severity following intervention. Positive reliable change was demonstrated for 6 of these 12 children, and one child showed negligible change. Two children showed an increase in ASD Index scores, reflecting an increase in ASD severity, and this increase was reliable deterioration for one of them. Two children showed no change in ASD severity from pre- to post- intervention. The ES was moderate for the GARS-3 ($d = -.56$) and statistically significantly greater than 0, 95% CI [-1.05, -.06].

After looking at the data points on the modified Brinley plot of the GARS-3, the data suggested that the children with lower scores on the ASD Index at pre-intervention seem to have shown the largest reduction in severity by post-intervention, whereas children with higher scores show less change. Therefore, a point biserial correlation was computed to determine whether treatment may be more effective among children with lower severity ratings on the GARS-3.

ASD severity was classified as low (< 108) or high (>108) using the pre-intervention GARS-3 scores. The results revealed that children who had a low severity rating pre-intervention had slightly better improvement in sleep outcomes (measured by the CSHQ) following treatment than those children who had a high severity rating pre-intervention ($r_{pb}(13) = -.39, p = .16$). Overall, 14.8% of the total variation in children's sleep difficulties measured by the CSHQ can be explained by the classification of low vs high severity on the GARS-3.

Modified Brinley Plots – Parent Outcomes

The RQI, PSQI, and the depression, anxiety and stress sub-scales scores of the DASS-21 are presented as modified Brinley plots showing individual change from pre- to post-intervention.

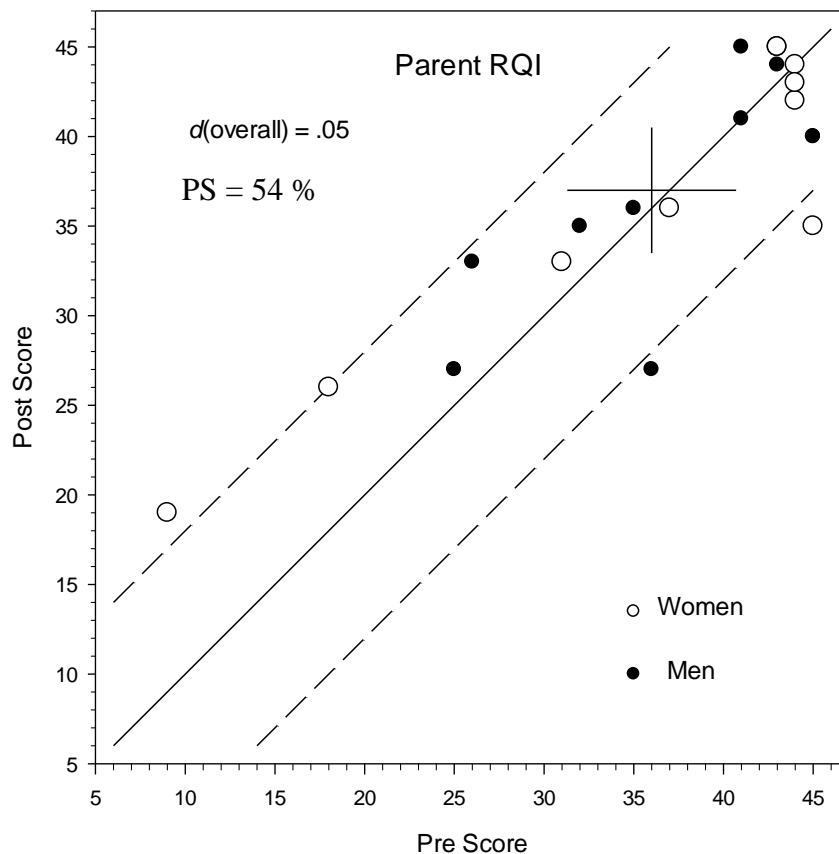


Figure 9. Modified Brinley plot showing change from pre-intervention to post-intervention on the RQI outcome measure for 18 parents who were in a relationship at the time of assessment. The cross marks the coordinate point of the means on each measure and the length of the cross arms show the 95 % confidence interval on the respective mean. d = Cohens d effect size; PS = Probability of Superiority.

RQI. As seen in Figure 9, 2 out of 18 participants showed no change in relationship quality between pre- and post-intervention. Ten out of 18 parents showed an improvement in relationship satisfaction, of these ten, two parents showed a significant improvement following intervention. Six out of 18 parents showed a decrease in RQI scores following

treatment, reflecting a decrease in relationship satisfaction. Of those who showed a decrease in RQI scores, two parents showed reliable deterioration. The ES was small for the RQI ($d = .05$) and not reliably different from 0, 95% CI [-0.20, .30].

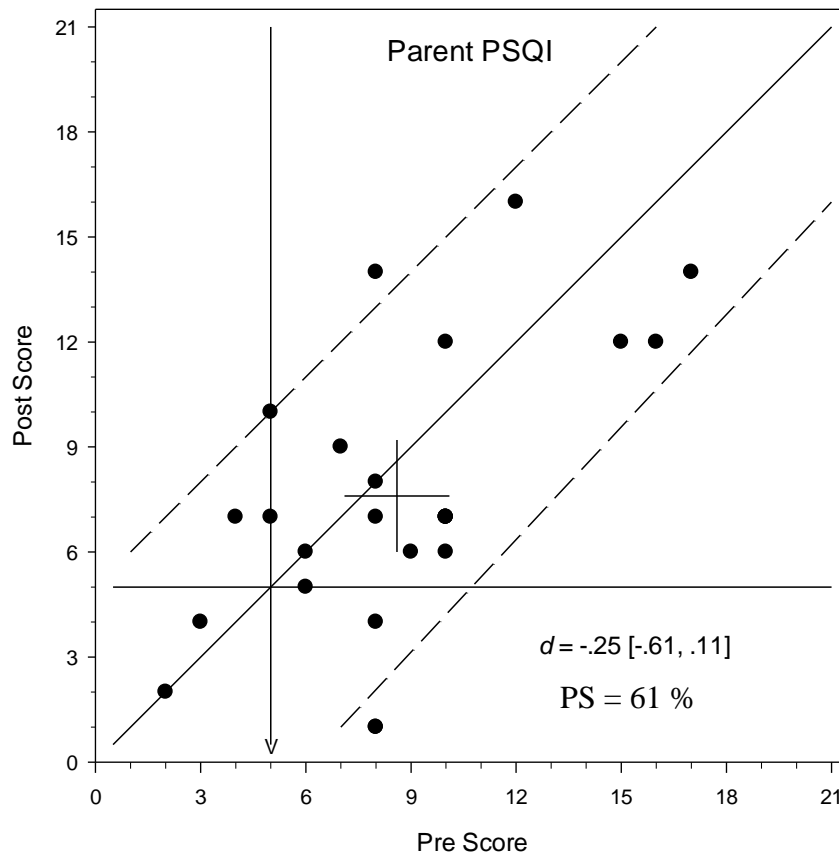


Figure 10. Modified Brinley plot showing change from pre-intervention to post-intervention on the PSQI outcome measure. Both vertical and horizontal lines are placed to show the clinical cut-off value. The arrowhead on the vertical cut-off indicates direction of desired change. The cross marks the coordinate point of the means on each measure and the length of the cross arms show the 95 % confidence interval on the respective mean. d = Cohens d effect size; PS = Probability of Superiority.

PSQI. As seen in Figure 10, 18 out of 25 parents had a PSQI score within the clinical range (between 5 and 21) pre-intervention. Six out of these 18 deteriorated post-intervention, with one of these parents showing a deterioration of sleep quality to a clinically significant degree. Ten parents showed an improvement in sleep quality, with two cases switching from the clinical to the non-clinical range and one improving to a clinically significant degree post-

intervention. Three parents were in the non-clinical range pre-intervention and this was maintained for two of these cases at post-intervention, with one of these cases deteriorating and moving from the non-clinical to the clinical range following treatment. Two parents showed no change from pre- to post intervention. The ES was small for the PSQI ($d = -.25$) and not statistically significantly different from 0, 95% CI [-.61, .11].

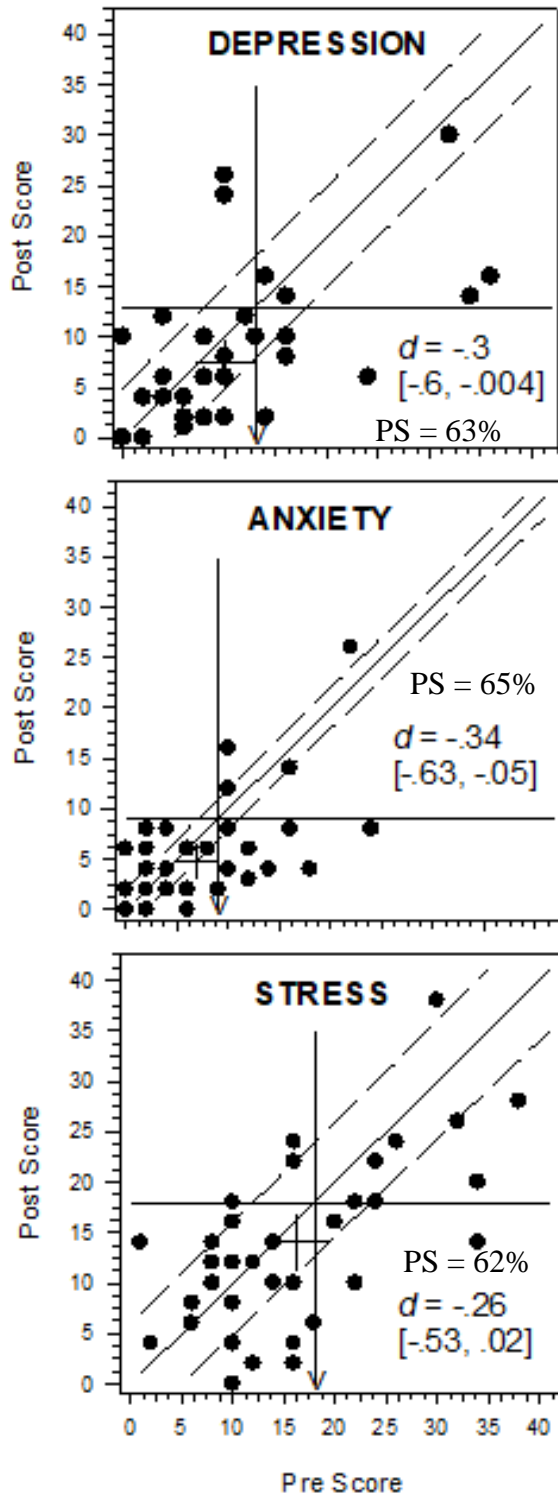


Figure 11. Modified Brinley plot showing change from pre- to post-intervention on the subscales of the DASS-21 outcome measure. Both vertical and horizontal lines are placed to show the clinical cut-off values. The arrowhead on the vertical cut-off indicates direction of desired change. The cross marks the coordinate point of the means on each measure and the length of the cross arms show the 95 % confidence interval on the respective mean. d = Cohens d effect size; PS = Probability of Superiority.

DASS-21. Consistent with the recommendation of Lovibond and Lovibond (1995) the DASS-21 raw scores were doubled so as to make them comparable with data from the full DASS.

Depression. As seen in Figure 11, 10 out of 38 parents had depression scores within the clinical range (mild-moderate boundary of > 14) pre-intervention. Of these parents, one showed an increase in depression levels while nine showed a reduction, with three cases showing clinically significant improvement post-treatment. Eighteen participants fell within the non-clinical range pre-intervention; of these participants, four cases showed reliable deterioration, and of these, two moved from the non-clinical to clinical range. The remainder of participants stayed within the non-clinical range post-intervention, with three participants showing negligible change. The ES was small for the Depression subscale on the DASS-21 ($d = -.30$) but since the 95% CI does not cross zero it is reliably non-0, 95% CI $[-.60, -.004]$.

Anxiety. As seen in Figure 11, 12 out of 38 participants were in the clinical range (>10) pre-intervention. Of these parents, two showed an increase in anxiety levels to the extent of reliable deterioration post-intervention, and eight participants moved from the clinical to the non-clinical range (mild-low anxiety range). Fifteen out of 38 participants were in the non-clinical range pre-intervention (mild to no anxiety) and this was maintained at post-intervention. Of these 15, two parent's anxiety scores showed reliable deterioration, four showed reliable improvement and four scores remained the same. The ES was small for the Anxiety sub-scale on the DASS-21 ($d = -.34$) but reliably non-0, 95% CI $[-.63, -.05]$.

Stress. As seen in Figure 11, 12 out of 38 participants had stress levels within the clinical range (mild to moderate boundary >19) pre-intervention. Of these parents, three showed a reduction in stress levels to the non-clinical range (mild to no stress), two of these

to a clinically significant degree. One case showed a reliable increase in stress level scores to a clinically significant degree. Twenty-one out of 38 participants were in the non-clinical range pre-intervention (mild to no stress). Of these parents, three showed an increase in stress levels moving from the non-clinical to the clinical range with one of these participants reporting increased stress to a clinically significant degree post-intervention. The remainder stayed within the non-clinical range with three parents showing no change. The ES was small for the stress subscale on the DASS-21 ($d = -.26$) and was not reliably different from 0, 95% CI [-.53, .02].

Treatment Acceptability- TARF-R

Table 7

Post-Intervention Treatment Acceptability Scores from TARF-R

TARF- R Subscale	Mean	SD	Range	Maximum Possible Score
Reasonableness	19.08	2.04	8	21
Effectiveness	18.21	3.51	15	21
Side Effects	16.81	4.76	18	21
Disruptive/Time	14.62	4.56	14	21
Cost	12.92	2.0	7	14
Willingness	18	3.05	13	21
Problem Severity	9.85	3.28	12	14
Understanding [°]	6.42	0.78	3	7
Total Acceptability [°]	100.27	15.56	69	119

Note. ° = not included in the total acceptability score

The TARF-R was completed by 17 of the participants. In seven cases, maternal and paternal ratings were provided. Table 7 displays the mean scores, standard deviation and range of the TARF-R for each parent post-intervention. The average overall acceptability rating for all participants was 100 out of 119. This suggests that overall; parents generally reported that interventions were effective, acceptable and clear to understand. Results suggested that all parents thought that the treatment they received was reasonable given the types of sleep problems with an average parent score of 19 out of 21. Similarly, fathers thought the treatment was more effective at solving the sleep issues than mothers (mothers: 18 out of 21, fathers: 20 out of 21). Results suggested that parents felt that much time was needed to carry out the intervention (mothers: 15 out of 21, fathers: 13 out of 21), that there was some cost involved in the treatment (mothers: 13 out of 14 fathers: 13 out of 14), some undesirable side effects (mothers: 16 out of 21, fathers: 17 out of 21) and on average parents understood the treatment process to a high degree (mothers: 6 out of 7, fathers: 6 out of 7).

Chapter 4

Discussion

This study indicates that there is promising evidence for the effectiveness of behavioural sleep interventions for children with ASD. The study did not find consistent improvements in secondary outcomes across all participants as measured by the CSHQ, GARS-3, CBCL, DASS-21, PSQI and RQI. However, the majority of participants improved on these measures and some to a clinically significant degree. The behavioural interventions were found to be effective in improving sleep and reducing ASD severity and problem behaviours (e.g., externalising behaviour) in children. Parental ratings of relationship satisfaction, sleep quality, depression, anxiety and stress also improved.

Research Questions

The aim of the present study was to examine whether improved sleep resulting from a behavioural sleep intervention affects children's daytime behaviour and parental well-being.

The following two research questions were posed:

1. Does a behavioural sleep intervention for children with ASD have any effect on their daytime functioning?
2. Does a behavioural sleep intervention for children have any effect on parental well-being in parents of a child with ASD?

Study Findings

The CSHQ (full and abbreviated version) were the primary outcome measures to assess change in sleep difficulties from pre- to post-intervention for children with ASD. The likelihood that a child had more sleep difficulties at pre-intervention than at post-intervention was 82%. Overall, in response to treatment, children's sleep showed improvement. The effect size was large (and significant); 11 children's sleep difficulties improved to a reliable extent; three children showed a small increase and one child showed little change following treatment.

Improvement in sleep could be attributed to the use of the FBA informed treatments in which interventions were individually tailored according to each child's specific needs, and the family environment. For the few children who deteriorated or showed no evidence of change, extraneous factors may have influenced sleep. For example, post-intervention data may have been collected at a time when there was a change to the family routine (e.g., school holidays, house relocation, birth of a sibling) or health (e.g., cold, flu) and we cannot exclude the possibility of parents finding the intervention process overwhelming and finding it hard to adhere to the treatment plan at that time.

Research Question One

Results of this study provide some evidence of the effectiveness of behavioural sleep interventions for reducing children's challenging daytime behaviour, including externalising behaviour problems, total problem behaviour (e.g., emotional reactivity, somatic complaints, attention problems and aggressive behaviour) and ASD severity. These perceived improvements were reflected in the CBCL and the GARS-3 scores for the children in the study.

The likelihood that a child had more externalising behaviour difficulties pre-intervention than at post-intervention was 83%. Thirteen out of the 17 parents reported an improvement in externalising behaviours, five of these children moved from the clinical to the non-clinical range and three children improved to a clinically significant degree. However, these improvements were not consistent across all participants. Two out of the 17 parents reported a small increase in their child's externalising behaviour problems and two out of 17 parents reported no change in externalising behaviour following intervention.

The likelihood that a child had more behaviour problems pre-intervention than at post-intervention was 63%. Eleven out of 17 the children's parents reported an improvement in total problem behaviours following treatment, four of these children improved and moved from the clinical to the non-clinical range and to a clinically significant degree. However, these improvements were not consistent across all participants. One out of 17 parents reported that their child's total problem behaviours remained the same following treatment and 5 out of the 17 parents reported that their children's total problem behaviours increased following treatment.

Recent studies exploring children's sleep difficulties and behaviour problems in children with ASD are mostly correlational, demonstrating a relationship between sleep difficulties and daytime functioning and behaviour (Allik et al., 2006; Bruni et al., 2007; Dahl & Harvey, 2007; DeVincent et al., 2007; Goldman et al., 2009; Henderson et al., 2011; Malow et al., 2006; Mayes & Calhoun, 2009; Moon et al., 2011; Patzold et al., 1998; Sikora et al., 2012). Very few studies have addressed whether improvement in children's sleep resulting from a successful behavioural sleep intervention had any effect on children's daytime behaviour. The findings of this study partially align with six case studies identified in the literature that found that behavioural sleep interventions had a positive effect on the daytime behaviours of children with ASD (e.g., externalising behaviour and SIB; (DeLeon et

al., 2004; Horner et al., 1997; Malow et al., 2014; Malow et al., 2006; Moon et al., 2011; Reed et al., 2009). It is noteworthy, however, that, as in the current study, the degree of change in daytime behaviour varied across studies and participants. Both previous correlational and intervention studies used a variety of measures to assess the effects of behavioural sleep interventions (e.g., actigraphy, CSHQ, sleep diaries, CBCL, Parent Satisfaction Questionnaire, Family Inventory of Sleep Habits, Parental Concerns Questionnaire, Parenting Stress Index) which may account for some of these differences.

The likelihood that a child had higher ASD severity rating pre-intervention than at post-intervention was 79%. Twelve out of 16 children's parents reported an improvement in their child's overall ASD symptomatology following treatment. However, these improvements were not consistent across all participants. Two out of 16 parents reported that their child's overall ASD symptomatology increased and one of these children showed reliable deterioration following treatment.

An interesting finding is a relationship between the GARS-3 severity scores pre-intervention and response to treatment. Children who were classified as having low GARS-3 severity scores at pre-intervention improved more substantially following treatment than those who had higher severity ratings. Such effects may be important in understanding the nature of sleep disturbances in children with more severe ASD symptomatology and may offer clues in understanding the factors relating to response to treatment. It is possible that sleep problems in children with severe ASD symptomatology as rated by the GARS-3 may have more of a physiological basis to their sleep problems; therefore, this is why fewer changes were seen in response to a behavioural sleep intervention.

Limited research has been conducted on the relationship between sleep and ASD symptomatology, with most studies being correlational. No studies have specifically looked

at whether successful treatment of sleep resulted in a reduction in ASD symptoms, however, the results of this study are consistent with Reed et al. (2009) who found improvements in ASD symptomatology measured by the Parental Concerns Questionnaire following a sleep intervention. Therefore; this research adds to the emerging research in this field and suggests promising effects for sleep interventions for reducing ASD severity and symptomology.

Overall, there are a number of possible reasons why improvements were observed in children's daytime functioning, behaviour and ASD symptomatology. For example, sleep difficulties and sleep deprivation may have similar origins, therefore changing the factor that influences one problem (e.g., sleep) may influence and change other problems (e.g., daytime behaviour) (Minde et al., 1994). Furthermore, factors maintaining sleep problems in children may also maintain daytime behaviour problems, therefore, parents who learn skills to manage problematic sleep behaviours, may generalise these skills and utilize them in other areas other than bedtime (Richdale & Wiggs, 2005). It is also likely that following a behavioural sleep intervention, well-being and mood improvements (e.g., depression, anxiety and stress) may have made parents more accepting of their child's daytime behaviour problems and ASD severity and rated it more favourably after intervention on the CBCL and GARS-3. In all behavioural interventions implemented in the study, bedtime routines and sleep hygiene were addressed. Mothers of children with sleep disturbance may have felt more in control of their child's behaviour at bedtime due to the installation of these routines and strategies to manage bedtime resistance. Therefore, this may explain the improvements seen in the parent's mood (e.g., depression, anxiety and stress). Similarly, a previous study has reported decreased parental stress with the installation of routines such as routines around dinner time, bedtime and carrying out chores (Fiese et al., 2002).

There are two plausible reasons why increases were observed in children's externalising behaviours, total problem behaviours and ASD severity ratings. The first is that

there may have been untreated co-occurring medical and behaviour comorbidities (e.g., sleep apnea, gastrointestinal problems and depression), rendering the sleep intervention less effective (Malow et al., 2014; Reynolds & Malow, 2011). The second is that problematic daytime behaviour may contribute to the sleep problem. Therefore daytime behaviour may not be significantly improved following a behavioural sleep intervention for children with ASD.

Research Question Two

Results of the study provide some evidence of the positive effects of behavioural sleep interventions on parents sleep quality (e.g., sleep duration, ability to fall asleep, sleep efficiency, level of sleep disturbance and daytime dysfunction), relationship satisfaction, depression, anxiety and stress levels. These perceived improvements and deteriorations were reflected in the PSQI, RQI and the DASS-21 scores for the parents of the children in this study.

The likelihood that a parent had lower sleep quality at pre-intervention than at post-intervention was 61%. Thirteen out of 25 parents reported an improvement in sleep quality with one parent improving to a clinically significant degree following treatment. However, these improvements were not consistent across all of the parents. Nine out of 25 parents reported that their sleep quality deteriorated; one parent moved from the non-clinical range to the clinical range and deteriorated to a clinically significant degree. Three parents reported no change following treatment of their child's sleep difficulties.

The current study is the first known study to look at the change in parental sleep quality following a behavioural sleep treatment in children with ASD. Previous research into the association between parent sleep quality and children's sleep difficulties is scarce, with only a few studies examining this relationship in TD children and children with intellectual

disabilities (Boergers et al., 2007; Robinson & Richdale, 2004; Wiggs & Stores, 2001). Only one study was found that investigated this relationship in children with ASD. Lopez-Wagner et al. (2008) showed positive correlations between sleep difficulties in children with ASD and poor parent sleep quality. However, this study was cross-sectional and could not address whether changes in children's sleep affected sleep quality in parents.

The likelihood that a parent had lower relationship satisfaction at pre-intervention than at post-intervention was 54%. Ten out of 18 parents reported an improvement in relationship satisfaction with two parents improving to a significant degree following treatment. However, these improvements were not consistent across all of the parents. Six out of the 18 parents reported that their relationship satisfaction deteriorated with one of these parents deteriorating to a significant degree. Two parents reported no change in relationship satisfaction following treatment of their child's sleep difficulties.

Before intervention, all parents had moderate to high scores on the RQI reflecting that parents were satisfied in their relationships. This is in contrast to previous research that suggests parents of children with ASD have elevated levels of marital dissatisfaction (Benson & Kersh, 2011; Lee, 2009; Lopez-Wagner et al., 2008). The current study is the first known study to examine the relationship satisfaction in parents following a behaviourally based sleep intervention for children with ASD. Additionally, no previous studies were found that looked at the relationship between marital satisfaction and sleep disturbances in children with ASD. However, this study found similar results to the intervention study conducted by Durand and Mindell (1990) which used modified extinction to treat sleep problems in TD children. Following intervention, they found improvements in both mothers and fathers satisfaction within their marriage (Durand & Mindell, 1990).

The likelihood that a parent had higher depression levels at pre-intervention than at post-intervention was 63%. Twenty-five out of 38 parents reported an improvement in depression levels with four parents improving to a clinically significant degree following treatment. However, these improvements were not consistent across all of the parents. Eight out of the 38 parents reported that their depression levels deteriorated with two of these parents deteriorating to a clinically significant degree moving from the non-clinical to the clinical range of depression. Five parents reported no change in depression levels following treatment of their child's sleep difficulties.

The likelihood that a parent had higher anxiety levels at pre-intervention than at post-intervention was 65%. Twenty out of 38 parents reported an improvement in anxiety levels, with 10 of these improving to a reliable extent and eight of these parents improving to a clinically significant degree following treatment. However, these improvements were not consistent across all of the parents. Ten out of the 38 parents reported that their anxiety levels deteriorated, four of these to a reliable extent and two of these parents deteriorating to a clinically significant degree moving from the non-clinical range to the clinical range of anxiety. Eight parents reported no change in anxiety levels following treatment of their child's sleep difficulties.

The likelihood that a parent had higher stress levels at pre-intervention than at post-intervention was 62%. Twenty out of 38 parents reported an improvement in stress levels, nine of these parents improved to a reliable extent with two parents improving to a clinically significant degree following treatment. However, these improvements were not consistently demonstrated across all of the parents. Fourteen out of the 38 parents reported that their stress levels deteriorated, four of these parents deteriorated to a reliable extent with two parents deteriorating to a clinically significant degree with one moving from the non-clinical range

into the clinical range following treatment. Four parents reported no change in stress levels following treatment of their child's sleep difficulties.

These results are in contrast to previous research that states that parents of children with sleep problems and ASD are at higher risk of being clinically depressed, anxious and stressed (Meltzer, 2011; Tilford et al., 2015). Benefits to parent wellbeing were achieved with a high understanding of treatment, which was reported to be effective, clear to understand and parents were highly satisfied with the intervention as a whole as reflected in the TARF-R scores.

This is the first known study to demonstrate a change in parent's well-being following a behaviourally based sleep intervention for children with ASD. Previous studies examining children's sleep and parent well-being have been cross-sectional (Chu & Richdale, 2009; Doo & Wing, 2006; Goodlin-Jones, Tang, et al., 2009; Hodge et al., 2013; Meltzer, 2011; Meltzer & Mindell, 2007; Shang et al., 2006; Stoléru et al., 1997). However, this study's findings are in line with previous research with TD children (Hauck et al., 2012; Hiscock et al., 2008; Lam et al., 2003; Mindell, Telofski, et al., 2009) and children with intellectual disabilities (Wiggs & Stores, 2001). These studies have found that interventions designed to improve sleep may also improve maternal mental health and stress in parents.

There are several possible reasons why improvements were observed in the parent's well-being following a child's behavioural sleep intervention. For example, parents may have increased self-efficacy and the skills needed to feel more confident about their parenting skills and the way they manage their children's behaviour around bedtime. Mothers in families with sleep problems are more likely to have doubts about their parenting competence and have difficulties setting limits (Morrell, 1999), therefore, behaviour sleep interventions may reduce stress in the home as well as increase feelings of hopefulness seeing an

improvement in their child's sleep. Secondly, even though the DASS-21 is not able to recognise the reasons for change, it is possible that depression, anxiety and stress scores decreased because of the parents receiving better quality and quantity of sleep.

Many of these parental wellbeing measures showed improvements as well as deteriorations in scores. Children's sleep problems may contribute to stress in mothers of children with ASD, but there may be other contributory factors that were untested and responsible for maternal stress including mothers own sleep difficulties and their children's ASD symptoms (Hoffman et al., 2008; Richdale, 2003) making it difficult to directly attribute the changes in parent well-being to the behavioural intervention alone. One possible explanation for either no change or a significant increase in depression, anxiety and stress levels in parents could be because maternal and paternal depression, anxiety and stress contribute to the sleep problem and therefore parental well-being may not be significantly improved following a behavioural sleep intervention for children with ASD. Additionally, children's behaviour problems may be linked to maternal mental health and not solely to their sleep difficulties (Allik et al., 2006).

One reason for the improvements seen in the parents sleep quality, as measured by the PSQI following intervention, could be because of improved sleep in children (e.g., reduction of nighttime awakenings and early morning waking's). It is quite possible for example, that a reduction in the frequency and duration of night wakings may have resulted in increased sleep quality and/or duration in parents. Children often awaken a parent during their night wakings which would likely impact the quality of parent sleep and subsequent daytime functioning (Meltzer & Montgomery-Downs, 2011). This is in line with evidence from a national survey of sleep in American children, which reported more than 50% of parents losing an average of 30 minutes of sleep per night due to their child's night awakenings (Meltzer & Montgomery-Downs, 2011).

The improvement seen in relationship satisfaction on the RQI could be attributed toward parents feeling as though they have worked as a team to resolve difficulties in their family life, boosting their confidence and overall satisfaction with one another. Deterioration or no improvement in parent's relationship satisfaction following a sleep intervention could be attributed to extraneous factors affecting family life including work stressors, health problems, financial strains as well as both parents not working as a team to implement treatment recommendations.

Lastly, greater reductions in children's sleep difficulties measured by the CSHQ from pre to post intervention correlated with greater changes in all secondary outcome measures at post-intervention. This finding demonstrated a change in the expected direction; however, the lack of significance for the correlations may be due to a lack of power related to the sample size.

The current study makes an important contribution to the scarce research investigating secondary outcomes following a behavioural sleep intervention for children with ASD. This study presents results on the secondary outcome measures of the CBCL, GARS-3, PSQI, RQI and DASS-21 from pre-to post-intervention. Following a sleep intervention, many participants improved to a clinically significant degree showing clinically significant improvements in children's externalising behaviour, total problem behaviours, parent's sleep quality, depression, anxiety and stress levels following treatment. However, it was found that some participants deteriorated to a clinically significant degree showing increases in externalising behaviour, total problem behaviours, ASD severity and the parent's depression, anxiety and stress levels following treatment.

Strengths of the study

This study contributes to the current field of literature by addressing many gaps. Currently, there is little research investigating the secondary outcomes of behaviourally-based sleep interventions, particularly for children with ASD. There is also very little research on the effect a child's behavioural sleep intervention has on parental well-being, especially relationship satisfaction. This study contributes to both of these gaps within the literature. The utilization of a range of psychometrics to assess parent well-being was also a strength of this study as it allowed multi-components of well-being (e.g., sleep quality, relationship satisfaction, depression, anxiety and stress) to be examined. Additionally, psychometrics were used with evidence of their reliability and validity for both parent and child measures. This study found that improved sleep can have wide-ranging effects on parental well-being as well as on children's behaviour. This study infers that sleep in children with ASD should be considered when other behavioural challenges and/or parents well-being is of concern.

Limitations

Five notable limitations are evident in this research study. The first limitation of the study is the reliance on parent report as a sole source of information about the child's sleep difficulties and all child and parent secondary outcomes. Psychometrics only measure the change in parent perceptions (Moore, 2004) and consequently, self-report measures can be highly vulnerable to response bias and inaccurate reporting of children's behaviour (Loring et al., 2018; Weiskop et al., 2005; Wilshire, 2017). This issue may have been further compounded by the fact that parents were not blind to the study or the intervention implemented. Whilst these limitations are common in the literature (Goodlin-Jones, Schwichtenberg, et al., 2009; Hering et al., 1999; Honomichl et al., 2002) it is important that they are given due consideration when interpreting these results.

A second limitation of the current study is that subjective reports of sleep difficulties, daytime functioning and parent well-being could be related to extraneous factors and not the result of a direct causal link. For example, confounding factors and participant differences such as job loss, relationship breakdowns family holidays and memory ability may have influenced parent's responses following treatment on the secondary outcome measures. These factors were not controlled for within the assessment process or data analysis.

A third limitation of the current study is the lack of follow-up data. There was a low response rate for long-term follow-up that was initially going to be completed. This data would have been beneficial to see if treatment effects and any change in secondary outcome measures were maintained long term.

The fourth limitation of this study is the change in the version of the CSHQ that was used. The full-scale CSHQ has established reliability and validity in typically developing children though there is less support for the abbreviated version of the CSHQ, especially in the ASD population (Owens et al., 2000). Furthermore, there was a lack of reference data for the abbreviated version of the CSHQ which prevented interpreting the modified Brinley plot with reference to clinical cut-offs. Additionally, Cohen's *d* and PS could not be calculated. The CSHQ abbreviated scoring form could not be obtained, therefore, a scoring form was adapted from the full-scale scoring form.

The fifth limitation of this study is the use of the CSHQ as the primary measure of sleep outcomes for children with ASD. Recent research undertaken by Johnson et al. (2016) found inconsistent internal consistency ratings across subtest of the CSHQ and they raised queries on the applicability of the subtests to individuals with ASD (Delemere & Dounavi, 2018). Therefore, the organisation and scoring structure for the CSHQ should be used with caution. Additionally, there is little psychometric information on the use and reliability of the

CSHQ in children with ASD (Adkins et al., 2012; Johnson et al., 2016; Rzepecka et al., 2011).

Future Directions

The findings and limitations of the current study lead to five recommendations for future research. Firstly, future research should use additional subjective measures other than those used in the current study. Subjective measures of sleep such as the use of behaviour and sleep diaries would be useful to track children's daytime behaviour (e.g., frequency charts of problem behaviour, teacher reports) and night-time sleep (e.g., sleep onset, sleep latency, night waking's). Future research should also include objective measures of sleep which will rely less on information from parents, therefore, preventing the chance of response bias and instead directly measuring sleep through technologies. Some of the most common include actigraphy, video-recording and polysomnography. Self-reported improvements would be strengthened with the evidence from objectively measured outcomes. Utilising these subjective and objective approaches to assess the effect of behavioural sleep interventions would likely provide better insight into secondary outcomes and provide a more rigorous and comprehensive research design (Moore, Evans, Hanvey, & Johnson, 2017).

Furthermore, the primary measure of sleep outcome in children was the CSHQ. Alternative tools might be considered to assess sleep behaviours and difficulties in children with ASD that have psychometric examination for use within the ASD population. This includes but not limited to the BEDS (Schreck et al., 2004) or the Modified Simonds and Parraga Sleep Questionnaire (MSPSQ) (Johnson et al., 2016; Simonds & Parraga, 1982).

Additionally, future work should include bigger populations and more participants. It is likely that the study resulted in some successful outcomes that are limited to the ASD

population. It would be beneficial to expand the current research to involve other developmental disabilities, ages and populations.

Future work should analyse the subscales of the GARS-3 (e.g., Restricted Repetitive Behaviours, Social Communication and Social Interaction) using modified Brinley plots. Past research has found correlational links between children's sleep difficulties and stereotypic and repetitive behaviours (Goldman et al., 2011; Schreck et al., 2004; Tudor et al., 2012). Therefore, it would have been beneficial to carry out this analysis to examine whether change was evident in these variables following a behavioural sleep intervention.

Lastly, future research should examine the sustainability of improved daytime functioning and parent well-being long-term. Future work should also include participant specific variables that correlate with response to intervention (e.g., ASD severity, age, number of diagnoses, gender, and maternal stress levels) and to establish which subscales (e.g., attention) were most sensitive to change following a behavioural sleep intervention.

Conclusion

This current study aimed to ascertain the effectiveness of behaviourally-based sleep interventions for children with ASD and any collateral benefit of reducing sleep problems. In conclusion, the current study provides evidence for the effectiveness of behavioural sleep interventions for these children. The study did not find consistent improvements across all participants, as measured by the CSHQ, GARS-3, CBCL, DASS-21, PSQI and RQI, however, the majority improved on these measures and some to a clinically significant degree. The behavioural interventions were found to be effective in improving sleep, relationship satisfaction, parent sleep quality as well as depression, anxiety and stress among parents and reducing ASD severity, problem behaviours (e.g., externalising behaviour) in children. These outcomes align with previously reported findings. Future research may

address the use of objective and subjective measures as well as participant specific variables in response to treatment.

References

- Achenbach, T. M., & Rescorla, L. A. (2000). Manual for the aseba school-age forms & profiles: An integrated system of mult-informant assessment. *Burlington: University of Vermont, Research Center for Children, Youth & Families.*
- Adair, R., Bauchner, H., Philipp, B., Levenson, S., & Zuckerman, B. (1991). Night waking during infancy: Role of parental presence at bedtime. *Pediatrics*, 87(4), 500.
- Adams, H., Matson, J. L., Cervantes, P., & Goldin, R. (2014). The relationship between autism symptom severity and sleep problems: Should bi-directionality be considered? *Research in Autism Spectrtrum Disorders*, 8(3), 193-199.
doi:10.1016/j.rasd.2013.11.008
- Adams, L. A., & Rickert, V. I. (1989). Reducing bedtime tantrums: Comparison between positive routines and graduated extinction. *Pediatrics*, 84(5), 756-761.
- Adkins, K. W., Molloy, C., Weiss, S. K., Reynolds, A., Goldman, S. E., Burnette, C., . . . Malow, B. A. (2012). Effects of a standardized pamphlet on insomnia in children with autism spectrum disorders. *Pediatrics*, 130 (Suppl 2), S139.
- Alfano, C. A. (2007). Sleep-related problems among children and adolescents with anxiety disorders. *J Am Acad Child Adolesc Psychiatry*, 46(2), 224-232.
doi:10.1097/01.chi.0000242233.06011.8e
- Allik, E., Larsson, J. O., & Smedje, H. (2008). Sleep patterns in school aged children with asperger syndrome or high functioning autism: A follow up study. *Journal of Autism and Developmental Disorders*, 38(9), 1625-1633. doi:10.1007/s10803-008-0543-0
- Allik, H., Larsson, J.-O., & Smedje, H. (2006). Insomnia in school-age children with asperger syndrome or high-functioning autism. *BMC psychiatry*, 6(1), 18-18.
doi:10.1186/1471-244X-6-18
- Altman, L., & Turk, J. M. (2016). Comparison of autistic profiles in young females and males with autism spectrum disorder: A review of the literature *Journal of the American Academy of Child & Adolescent Psychiatry*, 55(10), S106-S106.
doi:10.1016/j.jaac.2016.09.021
- Aman, M. G., Lam, K. S. L., & Collier-Crespin, A. (2003). Prevalence and patterns of use of psychoactive medicines among individuals with autism in the autism society of ohio. *Journal of Autism and Developmental Disorders*, 33(5), 527-534.
doi:10.1023/A:1025883612879

- American Academy of Sleep Medicine. (2005). The international classification of sleep disorders 2nd edition: Diagnostic and coding manual. *American Academy of Sleep Medicine W.*
- American Academy of Sleep Medicine. (2014). *International classification of sleep disorders: Diagnostic & coding manual, icsd-3*. Darien: American Academy of Sleep Medicine.
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*. American Psychiatric Association.
- Anders, T. F., Halpern, L. F., & Hua, J. (1992). Sleeping through the night: A developmental perspective. *Pediatrics*, 90(4), 554.
- Ando, H., & Yoshimura, I. (1979). Effects of age on communication skill levels and prevalence of maladaptive behaviors in autistic and mentally retarded children. *Journal of Autism and Developmental Disorders*, 9(1), 83-93.
- Angriman, M., Caravale, B., Novelli, L., Ferri, R., & Bruni, O. (2015). Sleep in children with neurodevelopmental disabilities. *Neuropediatrics*, 46(3), 199-210. doi:10.1055/s-0035-1550151
- Antony, M. M., Bieling, P. J., Cox, B. J., Enns, M. W., & Swinson, R. P. (1998). Psychometric properties of the 42-item and 21-item versions of the depression anxiety stress scales in clinical groups and a community sample. *Psychological Assessment*, 10(2), 176-181. doi:10.1037/1040-3590.10.2.176
- Arendt, J. (2005). Melatonin: Characteristics, concerns, and prospects. *Journal of Biological Rhythms*, 20(4), 291-303. doi:10.1177/0748730405277492
- Armstrong, K. L., Quinn, R. A., & Dadds, M. R. (1994). The sleep patterns of normal children. *The Medical Journal of Australia*, 161(3), 202-206.
- Atladóttir, H. Ó., Henriksen, T. B., Schendel, D. E., & Parner, E. T. (2012). Autism after infection, febrile episodes, and antibiotic use during pregnancy: An exploratory study. *Pediatrics*, 130(6), e1447-e1454. doi:10.1542/peds.2012-1107
- Atladóttir, H. Ó., Thorsen, P., Østergaard, L., Schendel, D. E., Lemcke, S., Abdallah, M., & Parner, E. T. (2010). Maternal infection requiring hospitalization during pregnancy and autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(12), 1423-1430.

- Attwood, T. (2007). *The complete guide to asperger's syndrome* (1st hardcover ed.). London;Philadelphia;: Jessica Kingsley Publishers.
- Autism Treatment Network Collaboration. (2010). Autism treatment network patient registry annual report. *Autism Speaks Foundation (Unpublished annual report)*.
- Backhaus, J., Junghanns, K., Broocks, A., Riemann, D., & Hohagen, F. (2002). Test–retest reliability and validity of the pittsburgh sleep quality index in primary insomnia. *Journal of Psychosomatic Research*, 53(3), 737-740. doi:10.1016/S0022-3999(02)00330-6
- Bailey, A., Le Couteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda, E., & Rutter, M. (1995). Autism as a strongly genetic disorder: Evidence from a british twin study. *Psychological medicine*, 25(1), 63-77.
- Baker, E. K., & Richdale, A. L. (2015). Sleep patterns in adults with a diagnosis of high-functioning autism spectrum disorder. *Sleep*, 38(11), 1765-1774. doi:10.5665/sleep.5160
- Bargiela, S., Steward, R., & Mandy, W. (2016). The experiences of late-diagnosed women with autism spectrum conditions: An investigation of the female autism phenotype. *Journal of Autism and Developmental Disorders*, 46(10), 3281-3294. doi:10.1007/s10803-016-2872-8
- Baron-Cohen, S., Cox, A., Baird, G., Swettenham, J., Nightingale, N., Morgan, K., . . . Charman, T. (1997). Psychological markers in the detection of autism in infancy in a large population. *Early Human Development*, 47(1), 98-99. doi:10.1016/S0378-3782(97)81293-5
- Bauman, M. L. (2010). Medical comorbidities in autism: Challenges to diagnosis and treatment. *Neurotherapeutics*, 7(3), 320-327. doi:10.1016/j.nurt.2010.06.001
- Bauman, M. L., & Kemper, T. L. (2005). *The neurobiology of autism* (2 ed.). Baltimore: Johns Hopkins University Press.
- Bauminger, N., Solomon, M., & Rogers, S. J. (2010). Externalizing and internalizing behaviors in asd. *Autism research*, 3(3), 101-112. doi:doi:10.1002/aur.131
- Bazil, C. W. (2003). Effects of antiepileptic drugs on sleep structure : Are all drugs equal? *CNS Drugs*, 17(10), 719-728.

- Begeer, S., Mandell, S., Wijnker-Holmes, B., Venderbosch, S., Rem, D., Stekelenburg, F., & Koot, H. M. (2013). Sex differences in the timing of identification among children and adults with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(5), 1151-1160. doi:10.1007/s10803-012-1656-z
- Bell, B. G., & Belsky, J. (2008). Parents, parenting, and children's sleep problems: Exploring reciprocal effects. *British Journal of Developmental Psychology*, 26(4), 579-593. doi:10.1348/026151008X285651
- Bellini, S. (2004). Social skill deficits and anxiety in high-functioning adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 19(2), 78-86. doi:10.1177/10883576040190020201
- Benson, B. A., & Brooks, W. T. (2008). Aggressive challenging behaviour and intellectual disability. *Current Opinion in Psychiatry*, 21(5), 454-458.
- Benson, P. R. (2006). The impact of child symptom severity on depressed mood among parents of children with asd: The mediating role of stress proliferation. *Journal of Autism and Developmental Disorders*, 36(5), 685-695. doi:10.1007/s10803-006-0112-3
- Benson, P. R., & Kersh, J. (2011). Marital quality and psychological adjustment among mothers of children with asd: Cross-sectional and longitudinal relationships. *Journal of Autism and Developmental Disorders*, 41(12), 1675-1685. doi:10.1007/s10803-011-1198-9
- Bierut, L. J., Hallmayer, J., Kim, C., Holt, R., Lotspeich, L., Gillberg, C., . . . Brennan, S. (2010). Functional impact of global rare copy number variation in autism spectrum disorders. *Nature*, 466(7304), 368-372. doi:10.1038/nature09146
- Bisulli, F., Vignatelli, L., Naldi, I., Pittau, F., Provini, F., Plazzi, G., . . . Tinuper, P. (2012). Diagnostic accuracy of a structured interview for nocturnal frontal lobe epilepsy (sinfle): A proposal for developing diagnostic criteria. *Sleep Medicine*, 13(1), 81-87. doi:10.1016/j.sleep.2011.09.003
- Blackmer, A. B., & Feinstein, J. A. (2016). Management of sleep disorders in children with neurodevelopmental disorders: A review. *Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy*, 36(1), 84-98. doi:10.1002/phar.1686
- Blampied, N. M. (2013). Functional behavioral analysis of sleep in infants and children. In *wolfson a. R., montgomery-downs h. E. (eds.), the oxford handbook of infant, child, and adolescent sleep and behavior* (pp. 169-188): New York, NY: Oxford University Press.

- Blampied, N. M. (2017). Analyzing therapeutic change using modified brinley plots: History, construction, and interpretation. *Behavior Therapy*, 48(1), 115-127.
- Blampied, N. M., & France, K. G. (1993). A behavioral model of infant sleep disturbance. *Journal of Applied Behavior Analysis*, 26(4), 477-492. doi:10.1901/jaba.1993.26-477
- Boergers, J., Hart, C., Owens, J. A., Streisand, R., & Spirito, A. (2007). Child sleep disorders: Associations with parental sleep duration and daytime sleepiness. *Journal of Family Psychology*, 21(1), 88-94. doi:10.1037/0893-3200.21.1.88
- Bonuck, K., & Grant, R. (2012). Sleep problems and early developmental delay: Implications for early intervention programs. *Intellectual and Developmental Disabilities*, 50(1), 41-52. doi:10.1352/1934-9556-50.1.41
- Brereton, A. V., Tonge, B. J., & Einfeld, S. L. (2006). Psychopathology in children and adolescents with autism compared to young people with intellectual disability. *Journal of Autism and Developmental Disorders*, 36(7), 863-870. doi:10.1007/s10803-006-0125-y
- Bristol, M. M., Gallagher, J. J., & Holt, K. D. (1993). Maternal depressive symptoms in autism: Response to psychoeducational intervention. *Rehabilitation Psychology*, 38(1), 3-10. doi:10.1037/h0080290
- Bromley, J., Hare, D. J., Davison, K., & Emerson, E. (2004). Mothers supporting children with autistic spectrum disorders: Social support, mental health status and satisfaction with services. *Autism*, 8(4), 409-423. doi:10.1177/1362361304047224
- Brown, A. S. (2012). Epidemiologic studies of exposure to prenatal infection and risk of schizophrenia and autism. *Developmental Neurobiology*, 72(10), 1272-1276. doi:10.1002/dneu.22024
- Brown, J. (2014). Autism spectrum disorder and sleep-related disturbances: A general overview. *Behavioral Health*, 1(2).
- Bruni, O., Ferri, R., Vittori, E., Novelli, L., Vignati, M., Porfirio, M. C., . . . Curatolo, P. (2007). Sleep architecture and nrem alterations in children and adolescents with asperger syndrome. *Sleep*, 30(11), 1577-1585. doi:10.1093/sleep/30.11.1577
- Buyse, D. J., Reynolds, C. F., 3rd, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Res*, 28(2), 193-213.

- Byars, K. C., Yolton, K., Rausch, J., Lanphear, B., & Beebe, D. W. (2012). Prevalence, patterns, and persistence of sleep problems in the first 3 years of life. *Pediatrics*, 129(2), e276-e284. doi:10.1542/peds.2011-0372
- Calhoun, S. L., Mayes, S. D., Vgontzas, A. N., Tsaoussoglou, M., Shifflett, L. J., & Bixler, E. O. (2009). No relationship between neurocognitive functioning and mild sleep disordered breathing in a community sample of children. *Journal of Clinical Sleep Medicine*, 5(3), 228-234.
- Camhi, S. L., Morgan, W. J., Pernisco, N., & Quan, S. F. (2000). Factors affecting sleep disturbances in children and adolescents. *Sleep Medicine*, 1(2), 117-123. doi:10.1016/S1389-9457(99)00005-2
- Careaga, M., Murai, T., & Bauman, M. D. (2017). Maternal immune activation and autism spectrum disorder: From rodents to nonhuman and human primates. *Biol Psychiatry*, 81(5), 391-401. doi:https://doi.org/10.1016/j.biopsych.2016.10.020
- Carpenter, J. S., & Andrykowski, M. A. (1998). Psychometric evaluation of the pittsburgh sleep quality index. *Journal of Psychosomatic Research*, 45(1), 5-13. doi:10.1016/S0022-3999(97)00298-5
- Carter, M., McCaughey, E., Annaz, D., & Hill, C. M. (2009). Sleep problems in a down syndrome population. *Archives of disease in childhood*, 94(4), 308-310. doi:10.1136/adc.2008.146845
- Carter, S. L. (2007). Review of recent treatment acceptability research. *Education and Training in Developmental Disabilities*, 42(3), 301-316.
- Cattaneo, L., Rizzolatti, G., & Fabbri-Destro, M. (2009). Mirror neurons and their clinical relevance. *Nature Clinical Practice Neurology*, 5(1), 24-34. doi:10.1038/ncpneuro0990
- Cavalieri, A. (2016). Sleep issues in children with autism spectrum disorder. *Pediatric Nursing*, 42(4), 169.
- Charman, T. (2002). The prevalence of autism spectrum disorders: Recent evidence and future challenges. *European Child & Adolescent Psychiatry*, 11(6), 249-256. doi:10.1007/s00787-002-0297-8
- Chaudhuri, S., & Chatterjee, N. (2015). Autism spectrum disorder: The present perspective. *Journal of College of Medical Sciences-Nepal*, 10(3), 37-47. doi:10.3126/jcmsn.v10i3.12775

- Chou, M.-C., Chou, W.-J., Chiang, H.-L., Wu, Y.-Y., Lee, J.-C., Wong, C.-C., & Gau, S. S.-F. (2012). Sleep problems among taiwanese children with autism, their siblings and typically developing children. *Research in Autism Spectrum Disorders*, 6(2), 665-672. doi:<https://doi.org/10.1016/j.rasd.2011.09.010>
- Christensen, D. L., Baio, J., Van Naarden Braun, K., Bilder, D., Charles, J., Constantino, J. N., . . . Prevention. (2016). Prevalence and characteristics of autism spectrum disorder among children aged 8 years-autism and developmental disabilities monitoring network, 11 sites, united states, 2012. *Morbidity and mortality weekly report. Surveillance summaries (Washington, D.C. : 2002)*, 65(3), 1.
- Christodulu, K. V., & Durand, V. M. (2004). Reducing bedtime disturbance and night waking using positive bedtime routines and sleep restriction. *Focus on Autism and Other Developmental Disabilities*, 19(3), 130-139.
- Chu, J., & Richdale, A. L. (2009). Sleep quality and psychological wellbeing in mothers of children with developmental disabilities. *Research in Developmental Disabilities*, 30(6), 1512-1522. doi:10.1016/j.ridd.2009.07.007
- Clara, I. P., Cox, B. J., & Enns, M. W. (2001). Confirmatory factor analysis of the depression–anxiety–stress scales in depressed and anxious patients. *Journal of Psychopathology and Behavioral Assessment*, 23(1), 61-67. doi:10.1023/A:1011095624717
- Clements, J., Wing, L., & Dunn, G. (1986). Sleep problems in handicapped children: A preliminary study. *Journal of Child Psychology and Psychiatry*, 27(3), 399-407. doi:10.1111/j.1469-7610.1986.tb01841.x
- Cohen-Ophir, M., Castel-Deutsh, T., & Tirosh, E. (2012). Autism in early childhood: An unusual developmental course; three case reports. *Case Reports in Psychiatry*, 2012, 4. doi:10.1155/2012/946109
- Cohen, I. L., Schmidt-Lackner, S., Romanczyk, R., & Sudhalter, V. (2003). The pdd behavior inventory: A rating scale for assessing response to intervention in children with pervasive developmental disorder. *Journal of Autism and Developmental Disorders*, 33(1), 31-45.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. 2nd: Hillsdale, NJ: erlbaum.
- Cohen, L. I., Yoo, J. H., Goodwin, M. S., & Moskowitz, L. (2011). Assessing challenging behaviors in autism spectrum disorders: Prevalence, rating scales, and autonomic indicators. In J. L. Matson & P. Sturmey (Eds.), *International handbook of autism and*

- pervasive developmental disorders* (pp. 247-270). New York, NY: Springer New York.
- Cohen, L. L., Feinstein, A., Masuda, A., & Vowles, K. E. (2014). Single-case research design in pediatric psychology: Considerations regarding data analysis. *Journal of Pediatric Psychology*, 39(2), 124-137. doi:10.1093/jpepsy/jst065
- Cohen, S., Conduit, R., Lockley, S. W., Rajaratnam, S. M., & Cornish, K. M. (2014). The relationship between sleep and behavior in autism spectrum disorder (asd): A review. *Journal of Neurodevelopmental Disorders*, 6(1), 44. doi:10.1186/1866-1955-6-44
- Conners, C. K. (2003). Conners 3rd edition. *Toronto, Ontario, Canada: MHS.*
- Connolly, J. J., & Hakonarson, H. (2014). Etiology of autism spectrum disorder: A genomics perspective. *Current Psychiatry Reports*, 16(11), 501. doi:10.1007/s11920-014-0501-9
- Conroy, M. A., Dunlap, G., Clarke, S., & Alter, P. J. (2005). A descriptive analysis of positive behavioral intervention research with young children with challenging behavior. *Topics in Early Childhood Special Education*, 25(3), 157-166. doi:10.1177/02711214050250030301
- Constantino, J. N. (2002). The social responsiveness scale. *Los Angeles, CA: Western Psychological Services.*
- Corkum, P., Tannock, R., & Moldofsky, H. (1998). Sleep disturbances in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 37(6), 637-646.
- Cortesi, F., Giannotti, F., Sebastiani, T., Panunzi, S., & Valente, D. (2012). Controlled-release melatonin, singly and combined with cognitive behavioural therapy for persistent insomnia in children with autism spectrum disorders: A randomised placebo control trial. *Journal of Sleep Research*, 21(6), 700-709. doi:10.1111/j.1365-2869.2012.01021.x
- Cotton, S., & Richdale, A. (2006). Brief report: Parental descriptions of sleep problems in children with autism, down syndrome, and prader-willi syndrome. *Research in Developmental Disabilities*, 27(2), 151-161. doi:10.1016/j.ridd.2004.12.003
- Couturier, J. L., Speechley, K. N., Steele, M., Norman, R., Stringer, B., & Nicolson, R. (2005). Parental perception of sleep problems in children of normal intelligence with pervasive developmental disorders: Prevalence, severity, and pattern. *Journal of the*

- American Academy of Child & Adolescent Psychiatry*, 44(8), 815-822.
doi:10.1097/01.chi.0000166377.22651.87
- Croen, L. A., Grether, J. K., Yoshida, C. K., Odouli, R., & Hendrick, V. (2011). Antidepressant use during pregnancy and childhood autism spectrum disorders. *Archives of general psychiatry*, 68(11), 1104-1112.
- Cumming, G. (2012). *Understanding the new statistics: Effect sizes, confidence intervals, and meta-analysis*. New York: Routledge.
- Dahl, R. E., & Harvey, A. G. (2007). Sleep in children and adolescents with behavioral and emotional disorders. *Sleep Medicine Clinics*, 2(3), 501-511.
doi:10.1016/j.jsmc.2007.05.002
- Davis, K. F., Parker, K. P., & Montgomery, G. L. (2004). Sleep in infants and young children: Part two: Common sleep problems. *Journal of Pediatric Health Care*, 18(3), 130-137. doi:10.1016/S0891-5245(03)00150-0
- Davis, N. O., & Carter, A. S. (2008). Parenting stress in mothers and fathers of toddlers with autism spectrum disorders: Associations with child characteristics. *Journal of Autism and Developmental Disorders*, 38(7), 1278-1291. doi:10.1007/s10803-007-0512-z
- de Miguel-Díez, J., Villa-Asensi, J. R., & Álvarez-Sala, J. L. (2003). Prevalence of sleep-disordered breathing in children with down syndrome: Polygraphic findings in 108 children. *Sleep*, 26(8), 1006-1009. doi:10.1093/sleep/26.8.1006
- Delahaye, J., Kovacs, E., Sikora, D., Hall, T. A., Orlich, F., Clemons, T. E., . . . Kuhlthau, K. (2014). The relationship between health-related quality of life and sleep problems in children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 8(3), 292-303. doi:10.1016/j.rasd.2013.12.015
- Delemere, E., & Dounavi, K. (2018). Parent-implemented bedtime fading and positive routines for children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 48(4), 1002-1019. doi:10.1007/s10803-017-3398-4
- DeLeon, I. G., Fisher, W. W., & Marhefka, J.-M. (2004). Decreasing self-injurious behavior associated with awakening in a child with autism and developmental delays. *Behavioral Interventions*, 19(2), 111-119.
- Deverman, B. E., & Patterson, P. H. (2009). Cytokines and cns development. *Neuron*, 64(1), 61-78. doi:10.1016/j.neuron.2009.09.002

- DeVincent, C. J., Gadow, K. D., Delosh, D., & Geller, L. (2007). Sleep disturbance and its relation to dsm-iv psychiatric symptoms in preschool-age children with pervasive developmental disorder and community controls. *Journal of Child Neurology*, 22(2), 161-169. doi:10.1177/0883073807300310
- Devnani, P. A., & Hegde, A. U. (2015). Autism and sleep disorders. *Journal of Pediatric Neurosciences*, 10(4), 304-307. doi:10.4103/1817-1745.174438
- Didden, R., Korzilius, H., Aperlo, B. v., Overloop, C. v., & Vries, M. d. (2002). Sleep problems and daytime problem behaviours in children with intellectual disability. *Journal of Intellectual Disability Research*, 46(7), 537-547. doi:10.1046/j.1365-2788.2002.00404.x
- Didden, R., & Sigafoos, J. (2001). A review of the nature and treatment of sleep disorders in individuals with developmental disabilities. *Research in Developmental Disabilities*, 22(4), 255-272. doi:10.1016/S0891-4222(01)00071-3
- Dillon, J. E., & Chervin, R. D. (2014). Chapter 15 - attention deficit, hyperactivity, and sleep disorders In R. Ferber, M. H. Kryger, & D. Gozal (Eds.), *Principles and practice of pediatric sleep medicine (second edition)* (pp. 111-124). Philadelphia: Content
- Dimitrov, D. M., & Rumrill, P. D. (2003). Pretest-posttest designs and measurement of change. *Work*, 20(2), 159-165.
- Diomedi, M., Curatolo, P., Scalise, A., Placidi, F., Caretto, F., & Gigli, G. L. (1999). Sleep abnormalities in mentally retarded autistic subjects: Down's syndrome with mental retardation and normal subjects. *Brain and Development*, 21(8), 548-553. doi:http://doi.org/10.1016/S0387-7604(99)00077-7
- Dodd, J., & Jackiewicz, T. (2015). The negotiations of involved fathers and intensive mothers around shared-bed sleeping with infants (co-sleeping). *Health Sociology Review*, 24(2), 213-225. doi:10.1080/14461242.2015.1032321
- Domingue, B. (2000). The experience of autism in the lives of families. *Autism spectrum disorders: A transactional developmental perspective. Communication and language intervention series*, 9, 369-393.
- Doo, S., & Wing, Y. K. (2006). Sleep problems of children with pervasive developmental disorders: Correlation with parental stress. *Dev Med Child Neurol*, 48(8), 650-655. doi:10.1017/s001216220600137x
- Doyen, C., Mighiu, D., Kaye, K., Colineaux, C., Beaumanoir, C., Mouraëff, Y., . . . Contejean, Y. (2011). Melatonin in children with autistic spectrum disorders: Recent

and practical data. *European Child & Adolescent Psychiatry*, 20(5), 231-239.
doi:10.1007/s00787-011-0162-8

Durand, V. M., & Christodulu, K. V. (2004). Description of a sleep-restriction program to reduce bedtime disturbances and night waking. *Journal of Positive Behavior Interventions*, 6(2), 83-91.

Durand, V. M., Gernert-Dott, P., & Mapstone, E. (1996). Treatment of sleep disorders in children with developmental disabilities. *Journal of the Association for Persons with Severe Handicaps*, 21(3), 114-122. doi:doi:10.1177/154079699602100302

Durand, V. M., & Mindell, J. A. (1999). Behavioral intervention for childhood sleep terrors. *Behavior Therapy*, 30(4), 705-715.

Durand, V. N., & Mindell, J. A. (1990). Behavioral treatment of multiple childhood sleep disorders: Effects on child and family. *Behavior Modification*, 14(1), 37-49.
doi:10.1177/01454455900141003

Dworzynski, K. P. D., Ronald, A. P. D., Bolton, P. P. D., & Happé, F. P. D. (2012). How different are girls and boys above and below the diagnostic threshold for autism spectrum disorders? *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(8), 788-797. doi:10.1016/j.jaac.2012.05.018

Eckerberg, B. (2002). Treatment of sleep problems in families with small children: Is written information enough? *Acta Paediatrica*, 91(8), 952-959.
doi:10.1080/080352502760148694

Eckerberg, B. (2004). Treatment of sleep problems in families with young children: Effects of treatment on family well-being. *Journal of American Academy Child and Adolescent Psychiatry*, 43(9), 1133.

Egger, G., Roetzer, K. M., Noor, A., Lionel, A. C., Mahmood, H., Schwarzbraun, T., . . . Vincent, J. B. (2014). Identification of risk genes for autism spectrum disorder through copy number variation analysis in austrian families. *neurogenetics*, 15(2), 117-127. doi:10.1007/s10048-014-0394-0

Elsabbagh, M., Divan, G., Koh, Y. J., Kim, Y. S., Kauchali, S., Marcín, C., . . . Fombonne, E. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism research*, 5(3), 160-179. doi:10.1002/aur.239

Emerson, E. (2001). *Challenging behaviour: Analysis and intervention in people with severe intellectual disabilities* (2nd ed.). Cambridge;New York;: Cambridge University Press.

- Emerson, E., Kiernan, C., Alborz, A., Reeves, D., Mason, L., Mason, H., . . . Hatton, C. (2001). The prevalence of challenging behaviors: A total population study. *Research in Developmental Disabilities*, 22(1), 77-93. doi:10.1016/S0891-4222(00)00061-5
- Enticott, P. G., Kennedy, H. A., Rinehart, N. J., Tonge, B. J., Bradshaw, J. L., Taffe, J. R., . . . Fitzgerald, P. B. (2012). Mirror neuron activity associated with social impairments but not age in autism spectrum disorder. *Biol Psychiatry*, 71(5), 427-433. doi:10.1016/j.biopsych.2011.09.001
- Estes, M. (2016). Pregnant woman's immune response could lead to brain disorders in kids. *NeuroscienceNews*. Retrieved from <http://neurosciencenews.com/immune-response-pregnancy-neurodevelopment-4966/>
- Estes, M. L., & McAllister, A. K. (2015). Immune mediators in the brain and peripheral tissues in autism spectrum disorder. *Nature Reviews Neuroscience*, 16(8), 469-486. doi:10.1038/nrn3978
- Fakhoury, M. (2015). Autistic spectrum disorders: A review of clinical features, theories and diagnosis. *International Journal of Developmental Neuroscience*, 43, 70-77. doi:10.1016/j.ijdevneu.2015.04.003
- Farmer, C., Butter, E., Mazurek, M. O., Cowan, C., Lainhart, J., Cook, E. H., . . . Aman, M. (2015). Aggression in children with autism spectrum disorders and a clinic-referred comparison group. *Autism*, 19(3), 281-291.
- Ferber, R. (1985). *Solving your child's sleep problems*: New York: Simon and Schuster.
- Ferber, R., & Kryger, M. H. (1995). *Principles and practice of sleep medicine in the child*. Philadelphia: Saunders.
- Fiese, B. H., Tomcho, T. J., Douglas, M., Josephs, K., Poltrock, S., & Baker, T. (2002). A review of 50 years of research on naturally occurring family routines and rituals: Cause for celebration? *Journal of Family Psychology*, 16(4), 381.
- Finn, C. A., & Sladeczek, I. E. (2001). Assessing the social validity of behavioral interventions: A review of treatment acceptability measures. *School Psychology Quarterly*, 16(2), 176-206. doi:10.1521/scpq.16.2.176.18703
- Fischbach, G. D., & Lord, C. (2010). The simons simplex collection: A resource for identification of autism genetic risk factors. *Neuron*, 68(2), 192-195. doi:10.1016/j.neuron.2010.10.006

- Fisher, J., Feekery, C., & Rowe, H. (2004). Treatment of maternal mood disorder and infant behaviour disturbance in an Australian private mothercraft unit: A follow-up study. *Archives of Women's Mental Health*, 7(1), 89-93. doi:10.1007/s00737-003-0041-5
- Fleischmann, A. (2004). Narratives published on the internet by parents of children with autism: What do they reveal and why is it important? *Focus on Autism and Other Developmental Disabilities*, 19(1), 35-43. doi:10.1177/10883576040190010501
- Folstein, S. E., & Rosen-Sheidley, B. (2001). Genetics of autism: Complex aetiology for a heterogeneous disorder. *Nature Reviews Genetics*, 2(12), 943-955. doi:10.1038/35103559
- France, K. G., & Blampied, N. M. (1999). Infant sleep disturbance: Description of a problem behaviour process. *Sleep Medicine Reviews*, 3(4), 265-280. doi:10.1053/smr.1999.0071
- France, K. G., & Blampied, N. M. (2005). Modifications of systematic ignoring in the management of infant sleep disturbance: Efficacy and infant distress. *Child & Family Behavior Therapy*, 27(1), 1-16. doi:10.1300/J019v27n01_01
- France, K. G., Henderson, J. M., & Hudson, S. M. (1996). Fact, act, and tact: A three-stage approach to treating the sleep problems of infants and young children. *Child and Adolescent Psychiatric Clinics*, 5(3), 581-600.
- Frazier, T. W. P., Georgiades, S. P., Bishop, S. L. P., & Hardan, A. Y. M. D. (2014). Behavioral and cognitive characteristics of females and males with autism in the Simons Simplex Collection. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(3), 329-340.e323. doi:10.1016/j.jaac.2013.12.004
- Gabriels, R. L., Cuccaro, M. L., Hill, D. E., Ivers, B. J., & Goldson, E. (2005). Repetitive behaviors in autism: Relationships with associated clinical features. *Research in Developmental Disabilities*, 26(2), 169-181. doi:10.1016/j.ridd.2004.05.003
- Gadow, K. D., DeVincent, C. J., Pomeroy, J., & Azizian, A. (2004). Psychiatric symptoms in preschool children with PDD and clinic and comparison samples. *Journal of Autism and Developmental Disorders*, 34(4), 379-393. doi:10.1023/B:JADD.0000037415.21458.93
- Gadow, K. D., DeVincent, C. J., Pomeroy, J., & Azizian, A. (2005). Comparison of DSM-IV symptoms in elementary school-age children with PDD versus clinic and community samples. *Autism*, 9(4), 392-415. doi:10.1177/1362361305056079

- Gadow, K. D., Guttman-Steinmetz, S., Rieffe, C., & DeVincent, C. J. (2012). Depression symptoms in boys with autism spectrum disorder and comparison samples. *Journal of Autism and Developmental Disorders*, 42(7), 1353-1363. doi:10.1007/s10803-011-1367-x
- Galland, B. C., & Mitchell, E. A. (2010). Helping children sleep. *Archives of disease in childhood*, 95(10), 850-853. doi:10.1136/adc.2009.162974
- Gallese, V. (2007). Before and below 'theory of mind': Embodied simulation and the neural correlates of social cognition. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 362(1480), 659-669. doi:10.1098/rstb.2006.2002
- Garay, P. A., Hsiao, E. Y., Patterson, P. H., & McAllister, A. K. (2013). Maternal immune activation causes age- and region-specific changes in brain cytokines in offspring throughout development. *Brain, behavior, and immunity*, 31, 54-68. doi:https://doi.org/10.1016/j.bbi.2012.07.008
- Gardener, H., Spiegelman, D., & Buka, S. L. (2009). Prenatal risk factors for autism: Comprehensive meta-analysis. *The British journal of psychiatry*, 195(1), 7-14.
- Gau, S. S.-F., Chou, M.-C., Lee, J.-C., Wong, C.-C., Chou, W.-J., Chen, M.-F., . . . Wu, Y.-Y. (2010). Behavioral problems and parenting style among taiwanese children with autism and their. *Psychiatry Clin Neurosci*, 64(1), 70-78. doi:10.1111/j.1440-1819.2009.02034.x
- Gavidia-Payne, S., Matthews, J., Hudson, A., Richdale, A., & Nankervis, K. (2003). *Stress in parents of children with disabilities: The parenting hassles scale*. Paper presented at the Helping families change conference, Sydney.
- Gaylor, E. E., Goodlin-Jones, B. L., & Anders, T. F. (2001). Classification of young children's sleep problems: A pilot study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 40(1), 61-67. doi:https://doi.org/10.1097/00004583-200101000-00017
- Georgiades, S., Szatmari, P., Duku, E., Zwaigenbaum, L., Bryson, S., Roberts, W., . . . Vaillancourt, T. (2011). Phenotypic overlap between core diagnostic features and emotional/behavioral problems in preschool children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41(10), 1321-1329.
- Giannotti, F., Cortesi, F., Cerquiglini, A., & Bernabei, P. (2006). An open-label study of controlled-release melatonin in treatment of sleep disorders in children with autism. *Journal of Autism and Developmental Disorders*, 36(6), 741-752. doi:10.1007/s10803-006-0116-z

- Giannotti, F., Cortesi, F., Cerquiglini, A., Miraglia, D., Vagnoni, C., Sebastiani, T., & Bernabei, P. (2008). An investigation of sleep characteristics, eeg abnormalities and epilepsy in developmentally regressed and non-regressed children with autism. *Journal Autism Developmental Disorders*, 38(10), 1888-1887. doi:10.1007/s10803-008-0584-4
- Giarelli, E., Wiggins, L. D., Rice, C. E., Levy, S. E., Kirby, R. S., Pinto-Martin, J., & Mandell, D. (2010). Sex differences in the evaluation and diagnosis of autism spectrum disorders among children. *Disability and Health Journal*, 3(2), 107-116. doi:10.1016/j.dhjo.2009.07.001
- Gilliam, J. E. (2016). Gilliam autism rating scale: 3rd edition. WARS.
- Gillott, A., Furniss, F., & Walter, A. (2001). Anxiety in high-functioning children with autism. *Autism*, 5(3), 277-286. doi:10.1177/1362361301005003005
- Glaze, D. G. (2004). Childhood insomnia: Why chris can't sleep. *Pediatric Clinics of North America*, 51(1), 33-50. doi:10.1016/S0031-3955(03)00176-7
- Glendinning, C. (1983). *Unshared care: Parents and their disabled children*. London: Routledge & K. Paul.
- Gloster, A. T., Rhoades, H. M., Novy, D., Klotsche, J., Senior, A., Kunik, M., . . . Stanley, M. A. (2008). Psychometric properties of the depression anxiety and stress scale-21 in older primary care patients. *Journal of Affective Disorders*, 110(3), 248-259. doi:10.1016/j.jad.2008.01.023
- Goldberg, W. A., & Keller, M. A. (2007). Co-sleeping during infancy and early childhood: Key findings and future directions. *Infant and Child Development*, 16(4), 457-469. doi:10.1002/icd.522
- Goldman, S. E., McGrew, S., Johnson, K. P., Richdale, A. L., Clemons, T., & Malow, B. A. (2011). Sleep is associated with problem behaviors in children and adolescents with autism spectrum disorders. *Research in Autism Spectrum Disorder*, 5(3) 1223-1229. doi:10.1016/j.rasd.2011.01.010
- Goldman, S. E., Richdale, A. L., Clemons, T., & Malow, B. A. (2012). Parental sleep concerns in autism spectrum disorders: Variations from childhood to adolescence. *Journal of Autism and Developmental Disorder*, 42(4) 531-538. doi:10.1007/s10803-011-1270-5

- Goldman, S. E., Surdyka, K., Cuevas, R., Adkins, K., Wang, L., & Malow, B. A. (2009). Defining the sleep phenotype in children with autism. *Developmental Neuropsychology*, 34(5), 560-573. doi:10.1080/87565640903133509
- Goodlin-Jones, B., Schwichtenberg, A. J., Iosif, A.-M., Tang, K., Liu, J., & Anders, T. F. (2009). Six-month persistence of sleep problems in young children with autism, developmental delay, and typical development. *Journal of the American Academy of Child & Adolescent Psychiatry*, 48(8), 847-854. doi:https://doi.org/10.1097/CHI.0b013e3181a8135a
- Goodlin-Jones, B., Tang, K., Liu, J., & Anders, T. F. (2009). Sleep problems, sleepiness and daytime behavior in preschool-age children. *Journal of Child Psychology and Psychiatry*, 50(12), 1532-1540 doi:10.1111/j.1469-7610.2009.02110.x
- Goodlin-Jones, B. L., Tang, K., Liu, J., & Anders, T. F. (2008). Sleep patterns in preschool-age children with autism, developmental delay and typical development. *Journal of the American Academy Child and Adolescent Psychiatry*, 47(8), 930-938. doi:10.1097/CHI.0b013e3181799f7c
- Gordon, H. A., Rucklidge, J. J., Blampied, N. M., & Johnstone, J. M. (2015). Clinically significant symptom reduction in children with attention-deficit/hyperactivity disorder treated with micronutrients: An open-label reversal design study. *Journal of Child and Adolescent Psychopharmacology*, 25(10), 783-798. doi:10.1089/cap.2015.0105
- Green, J., Gilchrist, A., Burton, D., & Cox, A. (2000). Social and psychiatric functioning in adolescents with asperger syndrome compared with conduct disorder. *Journal of Autism and Developmental Disorders*, 30(4), 279-293. doi:10.1023/a:1005523232106
- Gregory, A. M., & O'Connor, T. G. (2002). Sleep problems in childhood: A longitudinal study of developmental change and association with behavioral problems. *Journal of the American Academy of Child & Adolescent Psychiatry*, 41(8), 964-971. doi:10.1097/00004583-200208000-00015
- Griffith, G. M., Hastings, R. P., Nash, S., & Hill, C. (2010). Using matched groups to explore child behavior problems and maternal well-being in children with down syndrome and autism. *Journal of Autism and Developmental Disorders*, 40(5), 610-619. doi:10.1007/s10803-009-0906-1
- Guinchat, V., Thorsen, P., Laurent, C., Cans, C., Bodeau, N., & Cohen, D. (2012). Pre-, peri- and neonatal risk factors for autism. *Acta obstetricia et gynecologica Scandinavica*, 91(3), 287-300.

- Halladay, A. K., Bishop, S., Constantino, J. N., Daniels, A. M., Koenig, K., Palmer, K., . . . Szatmari, P. (2015). Sex and gender differences in autism spectrum disorder: Summarizing evidence gaps and identifying emerging areas of priority. *Molecular Autism*, 6(1), 36. doi:10.1186/s13229-015-0019-y
- Haney, J. L. (2016). Autism, females, and the dsm-5: Gender bias in autism diagnosis. *Social Work in Mental Health*, 14(4), 396-407. doi:10.1080/15332985.2015.1031858
- Hartley, S. L., Sikora, D. M., & McCoy, R. (2008). Prevalence and risk factors of maladaptive behaviour in young children with autistic disorder. *Journal of Intellectual Disability Research*, 52(10), 819-829. doi:10.1111/j.1365-2788.2008.01065.x
- Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy*, 40(8), 869-893.
- Hauck, Y. L., Hall, W. A., Dhaliwal, S. S., Bennett, E., & Wells, G. (2012). The effectiveness of an early parenting intervention for mothers with infants with sleep and settling concerns: A prospective non-equivalent before-after design. *Journal of Clinical Nursing*, 21(1-2), 52-62. doi:10.1111/j.1365-2702.2011.03734.x
- Henderson, J. A., Barry, T. D., Bader, S. H., & Jordan, S. S. (2011). The relation among sleep, routines, and externalizing behavior in children with an autism spectrum disorder. *Research in Autism Spectrum Disorders*, 5(2) 758-767. doi:10.1016/j.rasd.2010.09.003
- Henry, J. D., & Crawford, J. R. (2005). The short-form version of the depression anxiety stress scales (dass-21): Construct validity and normative data in a large non-clinical sample. *British Journal of Clinical Psychology*, 44(2), 227-239. doi:10.1348/014466505X29657
- Hering, E., Epstein, R., Elroy, S., Iancu, D., & Zelnik, N. (1999). Sleep patterns in autistic children. *Journal Autism Developmental Disorders*, 29(2) 143-147. doi:10.1023/a:1023092627223
- Hill, A. P., Zuckerman, K. E., Hagen, A. D., Kriz, D. J., Duvall, S. W., van Santen, J., . . . Fombonne, E. (2014). Aggressive behavior problems in children with autism spectrum disorders: Prevalence and correlates in a large clinical sample. *Research in Autism Spectrum Disorders*, 8(9), 1121-1133. doi:10.1016/j.rasd.2014.05.006
- Hiscock, H., Bayer, J. K., Hampton, A., Ukoumunne, O. C., & Wake, M. (2008). Long-term mother and child mental health effects of a population-based infant sleep intervention: Cluster-randomized, controlled trial. *Pediatrics*, 122(3), e621-e627. doi:10.1542/peds.2007-3783

- Hiscock, H., & Wake, M. (2002). Randomised controlled trial of behavioural infant sleep intervention to improve infant sleep and maternal mood. *Bmj*, 324(7345), 1062.
- Hodge, D., Carollo, T. M., Lewin, M., Hoffman, C. D., & Sweeney, D. P. (2014). Sleep patterns in children with and without autism spectrum disorders: Developmental comparisons. *Research in Developmental Disabilities*, 35(7), 1631-1638. doi:<https://doi.org/10.1016/j.ridd.2014.03.037>
- Hodge, D., Hoffman, C. D., Sweeney, D. P., & Riggs, M. L. (2013). Relationship between children's sleep and mental health in mothers of children with and without autism. *Journal of Autism and Developmental Disorders*, 43(4), 956. doi:10.1007/s10803-012-1639-0
- Hodge, D., Parnell, A. M. N., Hoffman, C. D., & Sweeney, D. P. (2012). Methods for assessing sleep in children with autism spectrum disorders: A review. *Research Autism Spectrum Disorders*, 6(4) 1337-1334. doi:10.1016/j.rasd.2012.05.009
- Hoffman, C. D., Sweeney, D. P., Gilliam, J. E., Apodaca, D. D., Lopez-Wagner, M. C., & Castillo, M. M. (2005). Sleep problems and symptomology in children with autism. *Focus on Autism and Other Developmental Disabilities*, 20(4), 194-200. doi:10.1177/10883576050200040101
- Hoffman, C. D., Sweeney, D. P., Gilliam, J. E., & Lopez-Wagner, M. C. (2006). Sleep problems in children with autism and in typically developing children. *Focus on Autism and Other Developmental Disabilities*, 21(3), 146-152. doi:10.1177/10883576060210030301
- Hoffman, C. D., Sweeney, D. P., Lopez-Wagner, M. C., Hodge, D., Nam, C. Y., & Botts, B. H. (2008). Children with autism: Sleep problems and mothers' stress. *Focus on Autism and Other Developmental Disabilities*, 23(3), 155-165. doi:10.1177/1088357608316271
- Hollway, J. A., & Aman, M. G. (2011). Sleep correlates of pervasive developmental disorders: A review of the literature. *Research in Developmental Disabilities*, 32. doi:10.1016/j.ridd.2011.04.001
- Hollway, J. A., Aman, M. G., & Butter, E. (2013). Correlates and risk markers for sleep disturbance in participants of the autism treatment network. *Journal of Autism and Developmental Disorders*, 43(12) 2830-2843. doi:10.1007/s10803-013-1830-y
- Honomichl, R. D., Goodlin-Jones, B. L., Burnham, M., Gaylor, E., & Anders, T. F. (2002). Sleep patterns of children with pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 32(6), 553-561. doi:10.1023/A:1021254914276

- Horner, R. H., Carr, E. G., Strain, P. S., Todd, A. W., & Reed, H. K. (2002). Problem behavior interventions for young children with autism: A research synthesis. *Journal of Autism and Developmental Disorders*, 32(5), 423-446. doi:10.1023/A:1020593922901
- Horner, R. H., Day, H. M., & Day, J. R. (1997). Using neutralizing routines to reduce problem behaviors. *Journal of Applied Behavior Analysis*, 30(4), 601-614. doi:10.1901/jaba.1997.30-601
- Horvath, K., Papadimitriou, J. C., Rabszty, A., Drachenberg, C., & Tildon, J. T. (1999). Gastrointestinal abnormalities in children with autistic disorder. *The Journal of pediatrics*, 135(5), 559-563. doi:10.1016/S0022-3476(99)70052-1
- Howerton, C. L., & Bale, T. L. (2012). Prenatal programming: At the intersection of maternal stress and immune activation. *Hormones and Behavior*, 62(3), 237-242. doi:https://doi.org/10.1016/j.yhbeh.2012.03.007
- Howlin, P., & Moore, A. (1997). Diagnosis in autism: A survey of over 1200 patients in the UK. *autism*, 1(2), 135-162.
- Huang, X.-n., Wang, H.-s., Zhang, L.-j., & Liu, X.-c. (2010). Co-sleeping and children's sleep in china. *Biological Rhythm Research*, 41(3), 169-181. doi:10.1080/09291011003687940
- Hundley, R. J., Shui, A., & Malow, B. A. (2016). Relationship between subtypes of restricted and repetitive behaviors and sleep disturbance in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(11), 3448-3457. doi:10.1007/s10803-016-2884-4
- Hunter, D. J. (2005). Gene-environment interactions in human diseases. *Nature Reviews Genetics*, 6(4), 287-298. doi:10.1038/nrg1578
- Hurtig, T., Kuusikko, S., Mattila, M.-L., Haapsamo, H., Ebeling, H., Jussila, K., . . . Moilanen, I. (2009). Multi-informant reports of psychiatric symptoms among high-functioning adolescents with asperger syndrome or autism. *Autism*, 13(6), 583-598. doi:10.1177/1362361309335719
- Idring, S., Rai, D., Dal, H., Dalman, C., Sturm, H., Zander, E., . . . Magnusson, C. (2012). Autism spectrum disorders in the stockholm youth cohort: Design, prevalence and validity. *PLoS ONE*, 7(7), e41280. doi:10.1371/journal.pone.0041280

- Jacobson, N. S., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology*, 59(1), 12.
- Jamain, S., Betancur, C., Quach, H., Philippe, A., Fellous, M., Giros, B., . . . Paris Autism Res Int Sibpair, P. (2002). Linkage and association of the glutamate receptor 6 gene with autism. *Molecular Psychiatry*, 7(3), 302-310. doi:10.1038/sj/mp/4000979
- Jan, J. E., Freeman, R. D., & Fast, D. K. (1999). Melatonin treatment of sleep-wake cycle disorders in children and adolescents. *Developmental Medicine and Child Neurology*, 41(7), 491-500. doi:10.1111/j.1469-8749.1999.tb00644.x
- Jan, J. E., & O'Donnell, M. E. (1996). Use of melatonin in the treatment of paediatric sleep disorders. *Journal of Pineal Research*, 21(4), 193-199. doi:doi:10.1111/j.1600-079X.1996.tb00286.x
- Jan, J. E., Owens, J. A., Weiss, M. D., Johnson, K. P., Wasdell, M. B., Freeman, R. D., & Ipsiroglu, O. S. (2008). Sleep hygiene for children with neurodevelopmental disabilities. *Pediatrics*, 122(6), 1343.
- Jang, J., Dixon, D. R., Tarbox, J., & Granpeesheh, D. (2011). Symptom severity and challenging behavior in children with asd. *Research in Autism Spectrum Disorders*, 5(3), 1028-1032. doi:10.1016/j.rasd.2010.11.008
- Jarrold, C., Boucher, J., & Smith, P. (1993). Symbolic play in autism: A review. *Journal of Autism and Developmental Disorders*, 23(2), 281-307. doi:10.1007/BF01046221
- Jiang, H.-y., Xu, L.-l., Shao, L., Xia, R.-m., Yu, Z.-h., Ling, Z.-x., . . . Ruan, B. (2016). Maternal infection during pregnancy and risk of autism spectrum disorders: A systematic review and meta-analysis. *Brain, behavior, and immunity*, 58, 165-172.
- Jin, C. S., Hanley, G. P., & Beaulieu, L. (2013). An individualized and comprehensive approach to treating sleep problems in young children. *Journal of Applied Behavior Analysis*, 46(1), 161. doi:10.1002/jaba.16
- Johns, M. W. (1991). A new method for measuring daytime sleepiness: The epworth sleepiness scale. *Sleep*, 14(6), 540-545.
- Johnson, C. R. (1996). Sleep problems in children with mental retardation and autism. *Child and Adolescent Psychiatric Clinics of North America*, 5(3), 673-683.

- Johnson, C. R., DeMand, A., Lecavalier, L., Smith, T., Aman, M., Foldes, E., & Scahill, L. (2016). Psychometric properties of the children's sleep habits questionnaire in children with autism spectrum disorder. *Sleep Medicine*, 20, 5-11. doi:<https://doi.org/10.1016/j.sleep.2015.12.005>
- Johnson, K. P., Giannotti, F., & Cortesi, F. (2009). Sleep patterns in autism spectrum disorders. *Child and Adolescent Psychiatric Clinics of North America*, 18(4), 917-928.
- Jones, D., & Verduyn, C. (1983). Behavioural management of sleep problems. *Archives of disease in childhood*, 58(6), 442-444.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous child*, 2(3), 217-250.
- Karimi, P., Kamali, E., Mousavi, S. M., & Karahmadi, M. (2017). Environmental factors influencing the risk of autism. *Journal of Research in Medical Sciences : The Official Journal of Isfahan University of Medical Sciences*, 22, 27. doi:10.4103/1735-1995.200272
- Karren, B. C. (2017). A test review: Gilliam, j. E. (2014). Gilliam autism rating scale—third edition (gars-3). *Journal of Psychoeducational Assessment*, 35(3), 342-346. doi:doi:10.1177/0734282916635465
- Karst, J. S., & Van Hecke, A. V. (2012). Parent and family impact of autism spectrum disorders: A review and proposed model for intervention evaluation. *Clinical Child and Family Psychology Review*, 15(3), 247-277. doi:10.1007/s10567-012-0119-6
- Keyes, K. M., Susser, E., Cheslack-postava, K., Fountain, C., Liu, K., & Bearman, P. S. (2012). Cohort effects explain the increase in autism diagnosis among children born from 1992 to 2003 in california. *International Journal of Epidemiology*, 41(2), 495-503. doi:10.1093/ije/dyr193
- Kim, J. A., Szatmari, P., Bryson, S. E., Streiner, D. L., & Wilson, F. J. (2000). The prevalence of anxiety and mood problems among children with autism and asperger syndrome. *Autism*, 4(2), 117-132. doi:10.1177/1362361300004002002
- Kim, Y. S., Kim, S.-J., Kim, Y.-K., Leventhal, B. L., Koh, Y.-J., Fombonne, E., . . . Grinker, R. R. (2011). Prevalence of autism spectrum disorders in a total population sample. *American Journal of Psychiatry*, 168(9), 904-912. doi:10.1176/appi.ajp.2011.10101532

- Kirkovski, M., Enticott, P. G., & Fitzgerald, P. B. (2013). A review of the role of female gender in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(11), 2584-2603. doi:10.1007/s10803-013-1811-1
- Kodak, T. P., & Piazza, C. C. P. (2008). Assessment and behavioral treatment of feeding and sleeping disorders in children with autism spectrum disorders. *Child and Adolescent Psychiatric Clinics of North America*, 17(4), 887-905. doi:10.1016/j.chc.2008.06.005
- Koegel, R. L., Koegel, R. L., Schreibman, L., Schreibman, L., Loos, L. M., Loos, L. M., . . . Plenis, A. J. (1992). Consistent stress profiles in mothers of children with autism. *Journal of Autism and Developmental Disorders*, 22(2), 205-216. doi:10.1007/BF01058151
- Kohlhuber, M., & Bolte, G. (2011). Influence of environmental noise on sleep quality and sleeping disorders-implications for health. *Bundesgesundheitsblatt, Gesundheitsforschung, Gesundheitsschutz*, 54(12), 1319-1324. doi:10.1007/s00103-011-1370-6
- Kolevzon, A., Gross, R., & Reichenberg, A. (2007). Prenatal and perinatal risk factors for autism: A review and integration of findings. *Archives of Pediatrics & Adolescent Medicine*, 161(4), 326-333. doi:10.1001/archpedi.161.4.326
- Konstantareas, M. M., & Homatidis, S. (1992). Mothers' and fathers' self-report of involvement with autistic, mentally delayed, and normal children. *Journal of Marriage and Family*, 54(1), 153-164.
- Köse, S., Yılmaz, H., Ocakoğlu, F. T., & Özbaran, N. B. (2017). Sleep problems in children with autism spectrum disorder and intellectual disability without autism spectrum disorder. *Sleep Medicine*, 40(Supplement C), 69-77. doi:https://doi.org/10.1016/j.sleep.2017.09.021
- Krakowiak, P., Goodlin-Jones, B., Hertz-Picciotto, I., Croen, L. A., & Hansen, R. L. (2008). Sleep problems in children with autism spectrum disorders, developmental delays, and typical development: A population-based study. *Journal of Sleep Research*, 17(2), 197-206. doi:10.1111/j.1365-2869.2008.00650.x
- Kulman, G., Lissoni, P., Rovelli, F., Roselli, M. G., Brivio, F., & Sequeri, P. (2000). Evidence of pineal endocrine hypofunction in autistic children. *Neuroendocrinology Letters*, 21(1), 31-34.
- Kuusikko, S., Pollock-Wurman, R., Jussila, K., Carter, A. S., Mattila, M.-L., Ebeling, H., . . . Moilanen, I. (2008). Social anxiety in high-functioning children and adolescents with

- autism and asperger syndrome. *Journal of Autism and Developmental Disorders*, 38(9), 1697-1709. doi:10.1007/s10803-008-0555-9
- Lai, M.-C., Lombardo, M. V., Pasco, G., Ruigrok, A. N. V., Wheelwright, S. J., Sadek, S. A., . . . Consortium, M. A. (2011). A behavioral comparison of male and female adults with high functioning autism spectrum conditions. *PLoS ONE*, 6(6), e20835. doi:10.1371/journal.pone.0020835
- Lai, M.-C. M. D. P., Lombardo, M. V. P., Auyeung, B. P., Chakrabarti, B. P., & Baron-Cohen, S. P. (2015). Sex/gender differences and autism: Setting the scene for future research. *Journal of the American Academy of Child & Adolescent Psychiatry*, 54(1), 11-24. doi:10.1016/j.jaac.2014.10.003
- Lai, M. C., Lombardo, M. V., & Baron-Cohen, S. (2014). Autism. *Lancet*, 383(9920), 896-910. doi:10.1016/S0140-6736(13)61539-1
- Lainhart, J. E. (1999). Psychiatric problems in individuals with autism, their parents and siblings. *International Review of Psychiatry*, 11(4), 278-298.
- Lakens, D. (2013). Calculating and reporting effect sizes to facilitate cumulative science: A practical primer for t-tests and anovas. *Frontiers in Psychology*, 4, 863. doi:10.3389/fpsyg.2013.00863
- Lam, P., Hiscock, H., & Wake, M. (2003). Outcomes of infant sleep problems: A longitudinal study of sleep, behavior, and maternal well-being. *Pediatrics*, 111(3), e203.
- Larsson, M., Weiss, B., Janson, S., Sundell, J., & Bornehag, C.-G. (2009). Associations between indoor environmental factors and parental-reported autistic spectrum disorders in children 6–8 years of age. *Neurotoxicology*, 30(5), 822-831. doi:10.1016/j.neuro.2009.01.011
- Lawton, C., France, K. G., & Blampied, N. M. (1991). Treatment of infant sleep disturbance by graduated extinction. *Child & Family Behavior Therapy*, 13(1), 39-56. doi:10.1300/J019v13n01_03
- LeBourgeois, M. K., Giannotti, F., Cortesi, F., Wolfson, A. R., & Harsh, J. (2005). The relationship between reported sleep quality and sleep hygiene in italian and american adolescents. *Pediatrics*, 115(1), 257-265. doi:10.1542/peds.2004-0815H
- Lecavalier, L. (2006). Behavioral and emotional problems in young people with pervasive developmental disorders: Relative prevalence, effects of subject characteristics, and

- empirical classification. *Journal of Autism and Developmental Disorders*, 36(8), 1101-1114.
- Lee, B. K., Magnusson, C., Gardner, R. M., Blomström, Å., Newschaffer, C. J., Burstyn, I., . . . Dalman, C. (2015). Maternal hospitalization with infection during pregnancy and risk of autism spectrum disorders. *Brain, behavior, and immunity*, 44, 100-105.
- Lee, C. Y. Q., Anderson, A., & Moore, D. W. (2014). Using video modeling to toilet train a child with autism. *Journal of Developmental and Physical Disabilities*, 26(2), 123-134. doi:10.1007/s10882-013-9348-y
- Lee, G. K. (2009). Parents of children with high functioning autism: How well do they cope and adjust? *Journal of Developmental and Physical Disabilities*, 21(2), 93-114. doi:10.1007/s10882-008-9128-2
- Leeson, R., Barbour, J., Romaniuk, D., & Warr, R. (1994). Management of infant sleep problems in a residential unit. *Child: Care, Health and Development*, 20(2), 89-100. doi:10.1111/j.1365-2214.1994.tb00856.x
- Levin, A., & Scher, A. (2016). Sleep problems in young children with autism spectrum disorders: A study of parenting stress, mothers' sleep-related cognitions, and bedtime behaviors. *CNS Neuroscience & Therapeutics*, 22(11), 921-927. doi:10.1111/cns.12651
- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., . . . Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36(7), 849-861. doi:10.1007/s10803-006-0123-0
- Li, S., Jin, X., Yan, C., Wu, S., Jiang, F., & Shen, X. (2009). Factors associated with bed and room sharing in chinese school-aged children. *Child: Care, Health and Development*, 35(2), 171-177. doi:10.1111/j.1365-2214.2008.00889.x
- Li, Y.-M., Ou, J.-J., Liu, L., Zhang, D., Zhao, J.-P., & Tang, S.-Y. (2016). Association between maternal obesity and autism spectrum disorder in offspring: A meta-analysis. *Journal of Autism and Developmental Disorders*, 46(1), 95-102. doi:10.1007/s10803-015-2549-8
- Lim, J., & Dinges, D. F. (2010). A meta-analysis of the impact of short-term sleep deprivation on cognitive variables. *Psychological Bulletin*, 136(3), 375-389. doi:10.1037/a0018883

- Liu, L., Zhang, D., Rodzinka-pasko, J. K., & Li, Y.-M. (2016). Environmental risk factors for autism spectrum disorders. *Der Nervenarzt*, 87(2), 55-61. doi:10.1007/s00115-016-0172-3
- Liu, X., Hubbard, J. A., Fabes, R. A., & Adam, J. B. (2006). Sleep disturbances and correlates of children with autism spectrum disorders. *Child Psychiatry and Human Development*, 37(2), 179-191. doi:10.1007/s10578-006-0028-3
- Lombardo, M. V., Moon, H. M., Su, J., Palmer, T. D., Courchesne, E., & Pramparo, T. (2017). Maternal immune activation dysregulation of the fetal brain transcriptome and relevance to the pathophysiology of autism spectrum disorder. *Molecular Psychiatry*, 23(4), 1001
- Loomes, R., Hull, L., & Mandy, W. P. L. (2017). What is the male-to-female ratio in autism spectrum disorder? A systematic review and meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry*, 56(6), 466-474. doi:https://doi.org/10.1016/j.jaac.2017.03.013
- Lopez-Wagner, M. C., Hoffman, C. D., Sweeney, D. P., Hodge, D., & Gilliam, J. E. (2008). Sleep problems of parents of typically developing children and parents of children with autism. *The Journal of Genetic Psychology*, 169(3), 245-259. doi:10.3200/GNTP.169.3.245-260
- Lord, C. (1995). Follow-up of two-year-olds referred for possible autism. *Journal of Child Psychology and Psychiatry*, 36(8), 1365-1382. doi:10.1111/j.1469-7610.1995.tb01669.x
- Loring, W. A., Johnston, R. L., Shui, A. M., & Malow, B. A. (2018). Impact of a brief behavioral intervention for insomnia on daytime behaviors in adolescents with autism spectrum disorders. *Journal of Contemporary Psychotherapy*, 1-13. doi:10.1007/s10879-018-9381-3
- Lotter, V. (1966). Epidemiology of autistic conditions in young children. *Social psychiatry*, 1(3), 124-135.
- Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the depression anxiety stress scales*. Sydney, N.S.W.: Psychology Foundation of Australia.
- Lyall, K., Schmidt, R. J., & Hertz-Picciotto, I. (2014). Maternal lifestyle and environmental risk factors for autism spectrum disorders. *International Journal of Epidemiology*, 43(2), 443-464. doi:10.1093/ije/dyt282

- Lyons, V., & Fitzgerald, M. (2007). Asperger (1906-1980) and kanner (1894-1981), the two pioneers of autism. *Journal of Autism and Developmental Disorders*, 37(10), 2022-2023. doi:10.1007/s10803-007-0383-3
- Magnuson, K. M., & Constantino, J. N. (2011). Characterization of depression in children with autism spectrum disorders. *Journal of Developmental and Behavioral Pediatrics : JDBP*, 32(4), 332-340. doi:10.1097/DBP.0b013e318213f56c
- Malkova, N. V., Collin, Z. Y., Hsiao, E. Y., Moore, M. J., & Patterson, P. H. (2012). Maternal immune activation yields offspring displaying mouse versions of the three core symptoms of autism. *Brain, behavior, and immunity*, 26(4), 607-616.
- Malow, B. (2004). Sleep disorders, epilepsy and autism. *Mental Retardation and Developmental Disabilities Research Reviews*, 10(2), 122-125. doi:10.1002/mrdd.20023
- Malow, B., Adkins, K., Reynolds, A. M., Weiss, S., Loh, A., Fawkes, D., . . . Clemons, T. (2014). Parent based sleep education for children with autism spectrum disorders. *Journal of Autism and Develop Disorders*, 44(1), 216-228. doi:10.1007/s10803-013-1866-z
- Malow, B., Adkins, K. W., McGrew, S. G., Wang, L., Goldman, S. E., Fawkes, D., & Burnette, C. (2012). Melatonin for sleep in children with autism: A controlled trial examining dose, tolerability, and outcomes. *Journal of Autism and Developmental Disorders*, 42(8), 1729-1737. doi:10.1007/s10803-011-1418-3
- Malow, B., Marzec, M., McGrew, S., Wang, L., Henderson, L., & Stone, W. (2006). Characterizing sleep in children with autism spectrum disorders: A multi-dimensional approach. *Sleep*, 29(12), 1563-1571.
- Malow, B. A., Byars, K., Johnson, K., Weiss, S., Bernal, P., Goldman, S. E., . . . Sleep Committee of the Autism Treatment, N. (2012). A practice pathway for the identification, evaluation, and management of insomnia in children and adolescents with autism spectrum disorders. *Pediatrics*, 130(2), S106-S124. doi:10.1542/peds.2012-0900I
- Mandy, W., Chilvers, R., Chowdhury, U., Salter, G., Seigal, A., & Skuse, D. (2012). Sex differences in autism spectrum disorder: Evidence from a large sample of children and adolescents. *Journal of Autism and Developmental Disorders*, 42(7), 1304-1313. doi:10.1007/s10803-011-1356-0
- Mannion, A., & Leader, G. (2013). An analysis of the predictors of comorbid psychopathology, gastrointestinal symptoms and epilepsy in children and adolescents

- with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 7(12), 1663-1671. doi:<https://doi.org/10.1016/j.rasd.2013.10.002>
- Mannion, A., Leader, G., & Healy, O. (2013a). An investigation of comorbid psychological disorders, sleep problems, gastrointestinal symptoms and epilepsy in children and adolescents with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 7(1), 35-42. doi:<http://dx.doi.org/10.1016/j.rasd.2012.05.002>
- Markov, D., Jaffe, F., & Doghramji, K. (2006). Update on parasomnias: A review for psychiatric practice. *Psychiatry (Edgmont)*, 3(7), 69-76.
- Martinez, J. R., Werch, B. L., & Conroy, M. A. (2016). School-based interventions targeting challenging behaviors exhibited by young children with autism spectrum disorder: A systematic literature review. *Education and Training in Autism and Developmental Disabilities*, 51(3), 265-280.
- Maski, K. P., Jeste, S. S., & Spence, S. J. (2011). Common neurological co-morbidities in autism spectrum disorders. *Current Opinion in Pediatrics*, 23(6), 609-615. doi:10.1097/MOP.0b013e32834c9282
- Matson, J. L., Ancona, M., & Wilkins, J. (2008). Sleep disturbances in adults with autism spectrum disorders and severe intellectual impairments. *Journal of Mental Health Research in Intellectual Disabilities*, 1(3) 129-139. doi:10.1080/19315860801988210
- Matson, J. L., & Kozlowski, A. M. (2011). The increasing prevalence of autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5(1), 418-425. doi:10.1016/j.rasd.2010.06.004
- Matson, J. L., & Nebel-Schwalm, M. (2007). Assessing challenging behaviors in children with autism spectrum disorders: A review. *Research in Developmental Disabilities*, 28(6), 567-579. doi:10.1016/j.ridd.2006.08.001
- Matson, J. L., Wilkins, J., & Macken, J. (2009). The relationship of challenging behaviours to severity and symptoms of autism spectrum disorders. *Journal of Mental Health Research in Intellectual Disabilities*, 2(1), 29-44. doi:10.1080/19315860802611415
- Mattila, M.-L., Hurtig, T., Haapsamo, H., Jussila, K., Kuusikko-Gauffin, S., Kielinen, M., . . . Moilanen, I. (2010). Comorbid psychiatric disorders associated with asperger syndrome/high-functioning autism: A community- and clinic-based study. *Journal of Autism and Developmental Disorders*, 40(9), 1080-1093. doi:10.1007/s10803-010-0958-2

- May, T., Cornish, K., Conduit, R., Rajaratnam, S. M. W., & Rinehart, N. J. (2015). Sleep in high-functioning children with autism: Longitudinal developmental change and associations with behavior problems. *Behavioral Sleep Medicine*, 13(1), 2-18. doi:10.1080/15402002.2013.829064
- Mayes, S. D., & Calhoun, S. L. (2009). Variables related to sleep problems in children with autism. *Res Autism Spectr Disord*, 3. doi:10.1016/j.rasd.2009.04.002
- Mayes, S. D., Calhoun, S. L., Bixler, E. O., & Vgontzas, A. N. (2008). Nonsignificance of sleep relative to iq and neuropsychological scores in predicting academic achievement. *Journal of Developmental and Behavioral Pediatrics*, 29(3), 206-212. doi:10.1097/DBP.0b013e31816d924f
- Mayes, S. D., Gorman, A. A., Hillwig-Garcia, J., & Syed, E. (2013). Suicide ideation and attempts in children with autism. *Research in Autism Spectrum Disorders*, 7(1), 109-119. doi:https://doi.org/10.1016/j.rasd.2012.07.009
- Mazefsky, C. A., Anderson, R., Conner, C. M., & Minshew, N. (2011). Child behavior checklist scores for school-aged children with autism: Preliminary evidence of patterns suggesting the need for referral. *Journal of Psychopathology and Behavioral Assessment*, 33(1), 31-37. doi:10.1007/s10862-010-9198-1
- Mazefsky, C. A., Conner, C. M., & Oswald, D. P. (2010). Association between depression and anxiety in high-functioning children with autism spectrum disorders and maternal mood symptoms. *Autism research*, 3(3), 120-127. doi:10.1002/aur.133
- Mazina, V., Gerdts, J., Trinh, S., Ankenman, K., Ward, T., Dennis, M. Y., . . . Bernier, R. (2015). Epigenetics of autism-related impairment: Copy number variation and maternal infection. *Journal of developmental and behavioral pediatrics : JDBP*, 36(2), 61. doi:10.1097/DBP.0000000000000126
- Mazurek, M. O., & Petroski, G. F. (2015). Sleep problems in children with autism spectrum disorder: Examining the contributions of sensory over-responsivity and anxiety. *Sleep Medicine*, 16(2), 270-279. doi:http://doi.org/10.1016/j.sleep.2014.11.006
- Mazurek, M. O., & Sohl, K. (2016). Sleep and behavioral problems in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(6), 1906-1915. doi:10.1007/s10803-016-2723-7
- McBean, A. L., McBean, A. L., & Schlosnagle, L. (2016). Sleep, health and memory: Comparing parents of typically developing children and parents of children with special health-care needs. *Journal of Sleep Research*, 25(1), 78-87. doi:10.1111/jsr.12329

- McClintock, K., Hall, S., & Oliver, C. (2003). Risk markers associated with challenging behaviours in people with intellectual disabilities: A meta-analytic study. *Journal of Intellectual Disability Research*, 47(6), 405-416. doi:10.1046/j.1365-2788.2003.00517.x
- McGraw, K. O., & Wong, S. P. (1992). A common language effect size statistic. *Psychological Bulletin*, 111(2), 361-365. doi:10.1037/0033-2909.111.2.361
- McKenna, J. J., & Volpe, L. E. (2007). Sleeping with baby: An internet-based sampling of parental experiences, choices, perceptions, and interpretations in a western industrialized context. *Infant and Child Development*, 16(4), 359-385. doi:10.1002/icd.525
- McLaughlin Crabtree, V., Beal Korhonen, J., Montgomery-Downs, H. E., Faye Jones, V., O'Brien, L. M., & Gozal, D. (2005). Cultural influences on the bedtime behaviors of young children. *Sleep Medicine*, 6(4), 319-324. doi:10.1016/j.sleep.2005.02.001
- McLay, L., Carnett, A., van der Meer, L., & Lang, R. (2015). Using a video modeling-based intervention package to toilet train two children with autism. *Journal of Developmental and Physical Disabilities*, 27(4), 431-451. doi:10.1007/s10882-015-9426-4
- McLay, L., France, K., Blampied, N., Danna, K., & Hunter, J. (2017). Using functional behavioral assessment to develop a multicomponent treatment for sleep problems in a 3-year-old boy with autism. *Clinical Case Studies*, 16(3), 254-270. doi:10.1177/1534650116688558
- McLennan, J. D., Lord, C., & Schopler, E. (1993). Sex differences in higher functioning people with autism. *Journal of Autism and Developmental Disorders*, 23(2), 217-228. doi:10.1007/BF01046216
- Melke, J., Goubran Botros, H., Chaste, P., Betancur, C., Nygren, G., Anckarsäter, H., . . . Lund, U. (2008). Abnormal melatonin synthesis in autism spectrum disorders. *Molecular Psychiatry*, 13(1), 90-98. doi:10.1038/sj.mp.4002016
- Meltzer, L. J. (2008). Brief report: Sleep in parents of children with autism spectrum disorders. *Journal of Pediatric Psychology*, 33(4), 380-386. doi:10.1093/jpepsy/jsn005
- Meltzer, L. J. (2010). Clinical management of behavioral insomnia of childhood: Treatment of bedtime problems and night wakings in young children. *Behavioral Sleep Medicine*, 8(3), 172-189. doi:10.1080/15402002.2010.487464

- Meltzer, L. J. (2011). Factors associated with depressive symptoms in parents of children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5(1), 361-367. doi:10.1016/j.rasd.2010.05.001
- Meltzer, L. J., & Mindell, J. A. (2007). Relationship between child sleep disturbances and maternal sleep, mood, and parenting stress: A pilot study. *Journal of Family Psychology*, 21(1), 67-73. doi:10.1037/0893-3200.21.1.67
- Meltzer, L. J., & Mindell, J. A. (2008). Behavioral sleep disorders in children and adolescents. *Sleep Medicine Clinic*, 37(8), 722-728. doi:10.1016/j.jsmc.2008.01.004
- Meltzer, L. J., & Mindell, J. A. (2014). Systematic review and meta-analysis of behavioral interventions for pediatric insomnia. *Journal of Pediatric Psychology*, 39(8), 932-948. doi:10.1093/jpepsy/jsu041
- Meltzer, L. J., & Montgomery-Downs, H. E. (2011). Sleep in the family. *Pediatric Clinics of North America*, 58(3), 765-774. doi:10.1016/j.pcl.2011.03.010
- Miller, C. B., Espie, C. A., Epstein, D. R., Friedman, L., Morin, C. M., Pigeon, W. R., . . . Kyle, S. D. (2014). The evidence base of sleep restriction therapy for treating insomnia disorder. *Sleep Medicine Reviews*, 18(5), 415-424. doi:https://doi.org/10.1016/j.smr.2014.01.006
- Minde, K., Faucon, A., & Falkner, S. (1994). Sleep problems in toddlers: Effects of treatment on their daytime behavior. *Journal of the American Academy of Child & Adolescent Psychiatry*, 33(8), 1114-1121. doi:10.1097/00004583-199410000-00007
- Mindell, J. A., & Durand, V. M. (1993). Treatment of childhood sleep disorders: Generalization across disorders and effects on family members. *Journal of Pediatric Psychology*, 18(6), 731-750. doi:10.1093/jpepsy/18.6.731
- Mindell, J. A., Kuhn, B., Lewin, D. S., Meltzer, L. J., & Sadeh, A. (2006). Behavioral treatment of bedtime problems and night wakings in infants and young children - an american academy of sleep medicine review. *Sleep*, 29(10), 1263-1276.
- Mindell, J. A., Leichman, E. S., Lee, C., Williamson, A. A., & Walters, R. M. (2017). Implementation of a nightly bedtime routine: How quickly do things improve? *Infant Behavior and Development*, 49, 220-227. doi:https://doi.org/10.1016/j.infbeh.2017.09.013
- Mindell, J. A., Li, A. M., Sadeh, A., Kwon, R., & Goh, D. Y. T. (2015). Bedtime routines for young children: A dose-dependent association with sleep outcomes. *Sleep*, 38(5), 717-722. doi:10.5665/sleep.4662

- Mindell, J. A., Meltzer, L. J., Carskadon, M. A., & Chervin, R. D. (2009a). Developmental aspects of sleep hygiene: Findings from the 2004 national sleep foundation sleep in america poll. *Sleep Medicine*, 10(7), 771-779.
- Mindell, J. A., Telofski, L. S., Wiegand, B., & Kurtz, E. S. (2009b). A nightly bedtime routine: Impact on sleep in young children and maternal mood. *Sleep*, 32(5), 599-606. doi:10.1093/sleep/32.5.599
- Minshawi, N. F., Hurwitz, S., Fodstad, J. C., Biebl, S., Morriss, D. H., & McDougale, C. J. (2014). The association between self-injurious behaviors and autism spectrum disorders. *Psychology research and behavior management*, 7, 125-136. doi:10.2147/PRBM.S44635
- Montes, G., & Halterman, J. S. (2007). Psychological functioning and coping among mothers of children with autism: A population-based study. *Pediatrics*, 119(5), e1040-e1046. doi:10.1542/peds.2006-2819
- Montgomery, P., Stores, G., & Wiggs, L. (2004). The relative efficacy of two brief treatments for sleep problems in young learning disabled (mentally retarded) children: A randomised controlled trial. *Archives of disease in childhood*, 89(2), 125-130. doi:10.1136/adc.2002.017202
- Moon, E. C., Corkum, P., & Smith, I. M. (2011). Case study: A case-series evaluation of a behavioral sleep intervention for three children with autism and primary insomnia. *Journal of Pediatric Psychology*, 36(1), 47-54. doi:10.1093/jpepsy/jsq057
- Moore, M. (2012). Behavioral sleep problems in children and adolescents. *Journal of Clinical Psychology in Medical Settings*, 19(1), 77-83. doi:10.1007/s10880-011-9282-z
- Moore, M., Evans, V., Hanvey, G., & Johnson, C. (2017). Assessment of sleep in children with autism spectrum disorder. *Children*, 4(8), 72. doi:10.3390/children4080072
- Moore, P. S. (2004). The use of social stories in a psychology service for children with learning disabilities: A case study of a sleep problem. *British Journal of Learning Disabilities*, 32(3), 133-138. doi:10.1111/j.1468-3156.2004.00278.x
- Morgenthaler, T. I., Owens, J., Alessi, C., Boehlecke, B., Brown, T. M., Coleman, J. J., . . . American Academy of Sleep, M. (2006). Practice parameters for behavioral treatment of bedtime problems and night wakings in infants and young children. *Sleep*, 29(10), 1277. doi:10.1093/sleep/29.10.1277

- Morrell, J. M. (1999). The role of maternal cognitions in infant sleep problems as assessed by a new instrument, the maternal cognitions about infant sleep questionnaire. *The Journal of Child Psychology and Psychiatry and Allied Disciplines*, 40(2), 247-258.
- Murata, E., Mohri, I., Kato-Nishimura, K., Iimura, J., Ogawa, M., Tachibana, M., . . . Taniike, M. (2017). Evaluation of behavioral change after adenotonsillectomy for obstructive sleep apnea in children with autism spectrum disorder. *Research in Developmental Disabilities*, 65, 127-139.
doi:<https://doi.org/10.1016/j.ridd.2017.04.012>
- Muratori, F., Narzisi, A., Tancredi, R., Cosenza, A., Calugi, S., Saviozzi, I., . . . Calderoni, S. (2011). The cbcl 1.5–5 and the identification of preschoolers with autism in Italy. *Epidemiology and Psychiatric Sciences*, 20(4), 329-338.
doi:10.1017/S204579601100045X
- Muris, P., Steerneman, P., Merckelbach, H., Holdrinet, I., & Meesters, C. (1998). Comorbid anxiety symptoms in children with pervasive developmental disorders. *Journal of anxiety disorders*, 12(4), 387-393.
- Murphy, O., Healy, O., & Leader, G. (2009). Risk factors for challenging behaviors among 157 children with autism spectrum disorder in Ireland. *Research in Autism Spectrum Disorders*, 3(2), 474-482.
- Mutluer, T., Karakoc Demirkaya, S., & Abali, O. (2016). Assessment of sleep problems and related risk factors observed in Turkish children with autism spectrum disorders. *Autism research*, 9(5), 536-542. doi:10.1002/aur.1542
- Nardone, S., Sharan Sams, D., Reuveni, E., Getselter, D., Oron, O., Karpuj, M., & Elliott, E. (2014). DNA methylation analysis of the autistic brain reveals multiple dysregulated biological pathways. *Translational Psychiatry*, 4, e433. doi:10.1038/tp.2014.70
- Nguyen, M., Roth, A., Kyzar, E. J., Poudel, M. K., Wong, K., Stewart, A. M., & Kalueff, A. V. (2014). Decoding the contribution of dopaminergic genes and pathways to autism spectrum disorder (ASD). *Neurochem International*, 66, 15-26.
doi:10.1016/j.neuint.2014.01.002
- Norton, R. (1983). Measuring marital quality: A critical look at the dependent variable. *Journal of Marriage and Family*, 45(1), 141-151.
- Nunes, M. L., & Bruni, O. (2015). Insomnia in childhood and adolescence: Clinical aspects, diagnosis, and therapeutic approach. *Jornal de Pediatria*, 91(6, Supplement 1), S26-S35. doi:<http://doi.org/10.1016/j.jped.2015.08.006>

- O'Connor, B. B., & Jenni, O. G. (2005). Children's sleep: An interplay between culture and biology. *Journal of American Academy of Child and Adolescent Psychiatry*, 44(8), 822.
- Oberman, L. M., Hubbard, E. M., McCleery, J. P., Altschuler, E. L., Ramachandran, V. S., & Pineda, J. A. (2005). Eeg evidence for mirror neuron dysfunction in autism spectrum disorders. *Cognitive Brain Research*, 24(2), 190-198.
doi:<https://doi.org/10.1016/j.cogbrainres.2005.01.014>
- Olsson, M. B., & Hwang, C. P. (2001). Depression in mothers and fathers of children with intellectual disability. *Journal of intellectual disability research*, 45(6), 535-543.
- Ooi, Y. P., Tan, Z. J., Lim, C. X., Goh, T. J., & Sung, M. (2011). Prevalence of behavioural and emotional problems in children with high-functioning autism spectrum disorders. *Australian and New Zealand Journal of Psychiatry*, 45(5), 370-375.
doi:10.3109/00048674.2010.534071
- Osborne, L. A., & Reed, P. (2008). Parents' perceptions of communication with professionals during the diagnosis of autism. *Autism*, 12(3), 309-324.
doi:10.1177/1362361307089517
- Ospina, M. B., Krebs Seida, J., Clark, B., Karkhaneh, M., Hartling, L., Tjosvold, L., . . . Smith, V. (2008). Behavioural and developmental interventions for autism spectrum disorder: A clinical systematic review. *PLoS ONE*, 3(11), e3755.
doi:10.1371/journal.pone.0003755
- Owens, J. A. (2017). Behavioural sleep problems in children. *UptoDate*.
- Owens, J. A., & Moore, M. (2017). Insomnia in infants and young children. *Pediatric Annals*, 46(9), E321-E326. doi:10.3928/19382359-20170816-02
- Owens, J. A., Spirito, A., & McGuinn, M. (2000). The children's sleep habits questionnaire (cshq): Psychometric properties of a survey instrument for school-aged children. *Sleep*, 23(8), 1043-1051. doi:10.1093/sleep/23.8.1d
- Owens, J. A., & Witmans, M. (2004). Sleep problems. *Current Problems in Pediatric and Adolescent Health Care*, 34(4), 154-179. doi:10.1016/j.cppeds.2003.10.003
- Owens, L. J., France, K. G., & Wiggs, L. (1999). Behavioural and cognitive-behavioural interventions for sleep disorders in infants and children: A review. *Sleep Medicine Reviews*, 3(4), 281-302. doi:10.1053/smr.1999.0082

- Ozonoff, S., Heung, K., Byrd, R., Hansen, R., & Hertz-Picciotto, I. (2008). The onset of autism: Patterns of symptom emergence in the first years of life. *Autism research*, 1(6), 320-328. doi:10.1002/aur.53
- Paavonen, E. J., Nieminen-von Wendt, T., Vanhala, R., Aronen, E. T., & Von Wendt, L. (2003). Effectiveness of melatonin in the treatment of sleep disturbances in children with asperger disorder. *Journal of Child and Adolescent Psychopharmacology*, 13(1), 83-95. doi:10.1089/104454603321666225
- Park, S., Cho, S.-C., Cho, I. H., Kim, B.-N., Kim, J.-W., Shin, M.-S., . . . Yoo, H. J. (2012). Sleep problems and their correlates and comorbid psychopathology of children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6(3), 1068-1072. doi:10.1016/j.rasd.2012.02.004
- Parker-Athill, E. C., & Tan, J. (2011). Maternal immune activation and autism spectrum disorder: Interleukin-6 signaling as a key mechanistic pathway. *NeuroSignals*, 18(2), 113-128. doi:10.1159/000319828
- Patel, M. R., Carr, J. E., Kim, C., Robles, A., & Eastridge, D. (2000). Functional analysis of aberrant behavior maintained by automatic reinforcement: Assessments of specific sensory reinforcers. *Research in Developmental Disabilities*, 21(5), 393-407.
- Patrick, J., Dyck, M., & Bramston, P. (2010). Depression anxiety stress scale: Is it valid for children and adolescents? *Journal of Clinical Psychology*, 66(9), 996-1007. doi:10.1002/jclp.20696
- Patterson, P. H., Xu, W., Smith, S. E., & Devarman, B. E. (2008). Maternal immune activation, cytokines and autism. *Autism*, (pp. 289-307): Springer.
- Patzold, L. M., Richdale, A. L., & Tonge, B. J. (1998). An investigation into sleep characteristics of children with autism and asperger's disorder. *Journal of Paediatrics and Child Health*, 34(6), 528-533. doi:10.1046/j.1440-1754.1998.00291.x
- Paval, D. (2017). A dopamine hypothesis of autism spectrum disorder. *Developmental Neuroscience*, 39(5), 355-360. doi:10.1159/000478725
- Persico, A. M., & Bourgeron, T. (2006). Searching for ways out of the autism maze: Genetic, epigenetic and environmental clues. *Trends in Neurosciences*, 29(7), 349-358. doi:https://doi.org/10.1016/j.tins.2006.05.010
- Piazza, C. C., Fisher, W. W., & Sherer, M. (1997). Treatment of multiple sleep problems in children with developmental disabilities: Faded bedtime with response cost versus

- bedtime scheduling. *Developmental Medicine and Child Neurology*, 39(6), 414-418. doi:10.1111/j.1469-8749.1997.tb07456.x
- Pisula, E. (2007). A comparative study of stress profiles in mothers of children with autism and those of children with down's syndrome. *Journal of Applied Research in Intellectual Disabilities*, 20(3), 274-278. doi:10.1111/j.1468-3148.2006.00342.x
- Polimeni, M. A., Richdale, A. L., & Francis, A. J. P. (2005). A survey of sleep problems in autism, asperger's disorder and typically developing children. *Journal of Intellectual Disability Research*, 49(4), 260-268. doi:10.1111/j.1365-2788.2005.00642.x
- Pritchard, A. A., & Appleton, P. (1988). Management of sleep problems in pre-school children: Effects of a behavioural programme on sleep routines, maternal depression and perceived control. *Early Child Development and Care*, 34(1), 227-240.
- Quine, L. (1991). Sleep problems in children with mental handicap. *Journal of Intellectual Disability Research*, 35(4), 269-290.
- Quine, L. Y. N., & Pahl, J. A. N. (1985). Examining the causes of stress in families with severely mentally handicapped children. *The British Journal of Social Work*, 15(5), 501-517.
- Rapin, I., & Katzman, R. (1998). Neurobiology of autism. *Annals of Neurology*, 43(1), 7-14. doi:10.1002/ana.410430106
- Rechtschaffen, A. (1998). Current perspectives on the function of sleep. *Perspectives in Biology and Medicine*, 41(3), 359-390. doi:10.1353/pbm.1998.0051
- Reed, H. E., McGrew, S. G., Artibee, K., Surdkya, K., Goldman, S. E., Frank, K., . . . Malow, B. A. (2009). Parent-based sleep education workshops in autism. *Journal of Child Neurology*, 24(8), 936-945. doi:10.1177/0883073808331348
- Reid, M. J., Walter, A. L., & O'Leary, S. G. (1999). Treatment of young children's bedtime refusal and nighttime wakings: A comparison of "standard" and graduated ignoring procedures. *Journal of Abnormal Child Psychology*, 27(1), 5-16. doi:10.1023/A:1022606206076
- Reimers, T. M., Wacker, D. P., Cooper, L. J., & Deraad, A. O. (1992). Acceptability of behavioural treatments for children- analog and naturalistic evaluations by parents. *School Psychology Review*, 21(4), 628-643.

- Reynolds, A. M., & Malow, B. A. (2011). Sleep and autism spectrum disorders. *Pediatric Clinics North America* 58(3), 685-698. doi:10.1016/j.pcl.2011.03.009
- Rezendes, D. L., & Scarpa, A. (2011). Associations between parental anxiety/depression and child behavior problems related to autism spectrum disorders: The roles of parenting stress and parenting self-efficacy. *Autism Research and Treatment*, 2011, 395190. doi:10.1155/2011/395190
- Richdale, A. (2003). A descriptive analysis of sleep behaviour in children with fragile x. *Journal of Intellectual and Developmental Disability*, 28(2), 135-144. doi:10.1080/1366825031000147076
- Richdale, A., & Wiggs, L. (2005). Behavioral approaches to the treatment of sleep problems in children with developmental disorders: What is the state of the art? *International Journal of Behavioral Consultation and Therapy*, 1(3), 165-190. doi:10.1037/h0100743
- Richdale, A., Francis, A., Gavidia-Payne, S., & Cotton, S. (2000). Stress, behaviour, and sleep problems in children with an intellectual disability. *Journal of Intellectual and Developmental Disability*, 25(2), 147-161. doi:10.1080/13269780050033562
- Richdale, A. L. (1992). *An investigation of circadian rhythms in childhood autism*. La Trobe University.
- Richdale, A. L. (1999). Sleep problems in autism: Prevalence, cause, and intervention. *Developmental Medicine and Child Neurology*, 41(1), 60-66. doi:10.1111/j.1469-8749.1999.tb00012.x
- Richdale, A. L. (2001). Sleep in children with autism and asperger syndrome. *Sleep Disturbance in Children and Adolescents with Disorders of Development: its Significance and Management*, 181-191.
- Richdale, A. L. (2013). Autism and other developmental disabilities. In Wolfson A. R., Montgomery-Down H. E. (Eds.), *The Oxford handbook of infant, child, and adolescent sleep and behavior*, 471-494. doi:10.1093/oxfordhb/9780199873630.013.0032
- Richdale, A. L., Baker, E., Short, M., & Gradisar, M. (2014). The role of insomnia, pre-sleep arousal and psychopathology symptoms in daytime impairment in adolescents with high functioning autism spectrum disorder. *Sleep Medicine*, 15(9), 1082-1088. doi:10.1016/j.sleep.2014.05.005

- Richdale, A. L., & Prior, M. R. (1995). The sleep/wake rhythm in children with autism. *European Child & Adolescent Psychiatry*, 4(3), 175.
- Richdale, A. L., & Schreck, K. A. (2009b). Sleep problems in autism spectrum disorders: Prevalence, nature, & possible biopsychosocial aetiologies. *Sleep Medicine Reviews*, 13(6), 403-411. doi:10.1016/j.smrv.2009.02.003
- Rizzolatti, G., & Fabbri-Destro, M. (2010). Mirror neurons: From discovery to autism. *Experimental Brain Research*, 200(3), 223-237. doi:10.1007/s00221-009-2002-3
- Roberts, S. (1993). Tackling sleep problems through clinic-based approach. *Health visitor*, 66(5), 173.
- Robinson, A. M., & Richdale, A. L. (2004). Sleep problems in children with an intellectual disability: Parental perceptions of sleep problems, and views of treatment effectiveness. *Child: Care, Health and Development*, 30(2), 139-150. doi:10.1111/j.1365-2214.2004.00395.x
- Robinson, E. B., Samocha, K. E., Kosmicki, J. A., McGrath, L., Neale, B. M., Perlis, R. H., & Daly, M. J. (2014). Autism spectrum disorder severity reflects the average contribution of de novo and familial influences. *Proceedings of the National Academy of Sciences*, 111(42), 15161.
- Rodriguez, A. J. (2007). Pediatric sleep and epilepsy. *Current Neurology and Neuroscience Reports*, 7(4), 342-347.
- Rolider, A., & Houten, R. V. (1984). Training parents to use extinction to eliminate nighttime crying by gradually increasing the criteria for ignoring crying. *Education and Treatment of Children*, 7(2), 119-124.
- Ronald, A., & Hoekstra, R. A. (2011). Autism spectrum disorders and autistic traits: A decade of new twin studies. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 156(3), 255-274. doi:10.1002/ajmg.b.31159
- Ronald, A., Plomin, R., & Happé, F. (2006). Time to give up on a single explanation for autism. *Nature Neuroscience*, 9(10), 1218-1220. doi:10.1038/nn1770
- Rotter, J. (1966). Generalised expectancies for internal versus external control of reinforcement. *Psychol Monographs*, 80, 609.

- Russell, E., & Sofronoff, K. (2005). Anxiety and social worries in children with asperger syndrome. *Australian & New Zealand Journal of Psychiatry*, 39(7), 633-638. doi:10.1080/j.1440-1614.2005.01637.x
- Rutter, M. (2000). Genetic studies of autism: From the 1970s into the millennium. *Journal of Abnormal Child Psychology*, 28(1), 3-14. doi:10.1023/A:1005113900068
- Rutter, M., Tizard, J., & Whitmore, K. (1970). Health, education, and behaviour. *London, ENG: Longmans Green*.
- Rzepecka, H., McKenzie, K., McClure, I., & Murphy, S. (2011). Sleep, anxiety and challenging behaviour in children with intellectual disability and/or autism spectrum disorder. *Research in Developmental Disabilities*, 32(6), 2758-2766. doi:10.1016/j.ridd.2011.05.034
- Sadeh, A. (2005). A brief screening questionnaire for infant sleep problems: Validation and findings for an internet sample. *Pediatrics*, 113(6), e570-e577
- Sadeh, A., Gruber, R., & Raviv, A. (2002). Sleep, neurobehavioral functioning, and behavior problems in school-age children. *Child Development*, 73(2), 405-417. doi:10.1111/1467-8624.00414
- Sadeh, A., Gruber, R., & Raviv, A. (2003). The effects of sleep restriction and extension on school-age children: What a difference an hour makes. *Child Development*, 74(2), 444-455. doi:10.1111/1467-8624.7402008
- Sadeh, A., Raviv, A., & Gruber, R. (2000). Sleep patterns and sleep disruptions in school-age children. *Developmental Psychology*, 36(3), 291-301. doi:10.1037/0012-1649.36.3.291
- Sadeh, A. V. I., Mindell, J. A., Luedtke, K., & Wiegand, B. (2009). Sleep and sleep ecology in the first 3 years: A web-based study. *Journal of Sleep Research*, 18(1), 60-73. doi:10.1111/j.1365-2869.2008.00699.x
- Saemundsen, E., Magnússon, P., Georgsdóttir, I., Egilsson, E., & Rafnsson, V. (2013). Prevalence of autism spectrum disorders in an icelandic birth cohort. *BMJ Open*, 3(6), e002748. doi:10.1136/bmjopen-2013-002748
- Sajith, S., & Clarke, D. (2007). Melatonin and sleep disorders associated with intellectual disability: A clinical review. *Journal of Intellectual Disabilities Res*, 51(1), 2-13. doi:10.1111/j.1365-2788.2006.00893.x

- Sanders, M. R., Turner, K. M. T., & Markie-Dadds, C. (2001). *Practitioners kit for standard triple p: Practitioner's manual for standard triple p*: Families International Publishing.
- Schaevitz, L. R., & Berger-Sweeney, J. E. (2012). Gene–environment interactions and epigenetic pathways in autism: The importance of one-carbon metabolism. *ILAR Journal*, 53(3-4), 322-340. doi:10.1093/ilar.53.3-4.322
- Scher, A., Hall, W. A., Zaidman-Zait, A., & Weinberg, J. (2010). Sleep quality, cortisol levels, and behavioral regulation in toddlers. *Developmental psychobiology*, 52(1), 44-n/a. doi:10.1002/dev.20410
- Scher, A., Tirosh, E., Jaffe, M., Rubin, L., Sadeh, A., & Lavie, P. (1995). Sleep patterns of infants and young children in israel. *International Journal of Behavioral Development*, 18(4), 701-711. doi:10.1177/016502549501800407
- Schoen, S. A., Miller, L. J., Brett-Green, B. A., & Nielsen, D. M. (2009). Physiological and behavioral differences in sensory processing: A comparison of children with autism spectrum disorder and sensory modulation disorder. *Frontiers in Integrative Neuroscience*, 3, 29. doi:10.3389/neuro.07.029.2009
- Schreck, K., Mulick, J. A., & Smith, A. F. (2004). Sleep problems as possible predictors of intensified symptoms of autism. *Research in Developmental Disabilities*, 25(1), 57-66. doi:10.1016/j.ridd.2003.04.007
- Schreck, K. A. (2001). Behavioral treatments for sleep problems in autism: Empirically supported or just universally accepted? *Behavioral Interventions*, 16(4), 265-278. doi:10.1002/bin.98
- Schreck, K. A., & Mulick, J. A. (2000). Parental report of sleep problems in children with autism. *Journal of Autism and Developmental Disorders*, 30(2), 127-135. doi:10.1023/a:1005407622050
- Schwichtenberg, A. J., Young, G. S., Hutman, T., Iosif, A. M., Sigman, M., Rogers, S. J., & Ozonoff, S. (2013). Behavior and sleep problems in children with a family history of autism. *Autism Research*, 6(3), 169-176. doi:10.1002/aur.1278
- Seltzer, M. M., Greenberg, J. S., & Krauss, M. W. (1995). A comparison of coping strategies of aging mothers of adults with mental illness or mental retardation. *Psychology and Aging*, 10(1), 64-75. doi:10.1037/0882-7974.10.1.64

- Shang, C. Y., Gau, S. S. F., & Soong, W. T. (2006). Association between childhood sleep problems and perinatal factors, parental mental distress and behavioral problems. *Journal of Sleep Research, 15*(1), 63-73. doi:10.1111/j.1365-2869.2006.00492.x
- Sharma, M. P., & Andrade, C. (2012). Behavioral interventions for insomnia: Theory and practice. *Indian Journal of Psychiatry, 54*(4), 359-366. doi:10.4103/0019-5545.104825
- Shi, L., Fatemi, S. H., Sidwell, R. W., & Patterson, P. H. (2003). Maternal influenza infection causes marked behavioral and pharmacological changes in the offspring. *Journal of Neuroscience, 23*(1), 297-302.
- Sigafoos, J., O'Reilly, M. F., & Arthur-Kelly, M. (2003). *Challenging behavior and developmental disability*. London: Whurr.
- Sikora, D. M., Johnson, K., Clemons, T., & Katz, T. (2012). The relationship between sleep problems and daytime behavior in children of different ages with autism spectrum disorders. *Pediatrics, 130*(2), 83-90. doi:10.1542/peds.2012-0900F
- Simakajornboon, N., Kheirandish-Gozal, L., & Gozal, D. (2009). Diagnosis and management of restless legs syndrome in children. *Sleep Medicine Reviews, 13*(2), 149-156. doi:10.1016/j.smr.2008.12.002
- Simonds, J. F., & Parraga, H. (1982). Prevalence of sleep disorders and sleep behaviors in children and adolescents. *Journal of the American Academy of Child Psychiatry, 21*(4), 383-388. doi:https://doi.org/10.1016/S0002-7138(09)60942-0
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child & Adolescent Psychiatry, 47*(8), 921-929.
- Singh, K., & Zimmerman, A. W. (2015). Sleep in autism spectrum disorder and attention deficit hyperactivity disorder. *Seminars in pediatric neurology, 22*(2), 113-125. doi:10.1016/j.spen.2015.03.006
- Sivertsen, B., Posserud, M. B., Gillberg, C., Lundervold, A. J., & Hysing, M. (2012). Sleep problems in children with autism spectrum problems: A longitudinal population-based study. *Autism, 16*(2), 139-150. doi:10.1177/1362361311404255
- Skinner, B. F. (1969). *Contingencies of reinforcement: A theoretical analysis*. New York: Appleton-Century-Crofts.

- Sloper, P., Knussen, C., Turner, S., & Cunningham, C. (1991). Factors related to stress and satisfaction with life in families of children with down's syndrome. *Journal of Child Psychology and Psychiatry*, 32(4), 655-676. doi:10.1111/j.1469-7610.1991.tb00342.x
- Smith, S. E., Li, J., Garbett, K., Mirnics, K., & Patterson, P. H. (2007). Maternal immune activation alters fetal brain development through interleukin-6. *Journal of Neuroscience*, 27(40), 10695-10702.
- Smolders, S., Smolders, S. M. T., Swinnen, N., Gärtner, A., Rigo, J.-M., Legendre, P., & Brône, B. (2015). Maternal immune activation evoked by polyinosinic:Polycytidylic acid does not evoke microglial cell activation in the embryo. *Frontiers in Cellular Neuroscience*, 9(301). doi:10.3389/fncel.2015.00301
- Soke, G. N., Rosenberg, S. A., Hamman, R. F., Fingerlin, T., Rosenberg, C. R., Carpenter, L., . . . DiGuseppi, C. (2017). Factors associated with self-injurious behaviors in children with autism spectrum disorder: Findings from two large national samples. *Journal of Autism and Developmental Disorders*, 47(2), 285-296. doi:10.1007/s10803-016-2951-x
- Solomon, M., Miller, M., Taylor, S. L., Hinshaw, S. P., & Carter, C. S. (2012). Autism symptoms and internalizing psychopathology in girls and boys with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(1), 48-59. doi:10.1007/s10803-011-1215-z
- Souders, M. C., Mason, T. B., Valladares, O., Bucan, M., Levy, S. E., Mandell, D., . . . Pinto-Martin, J. (2009). Sleep behaviors and sleep quality in children with autism spectrum disorders. *Sleep*, 32(12), 1566-1578.
- Spence, S. H. (1998). A measure of anxiety symptoms among children. *Behaviour Research and Therapy*, 36(5), 545-566. doi:10.1016/S0005-7967(98)00034-5
- Spruyt, K., & Curfs, L. M. G. (2015). Non-pharmacological management of problematic sleeping in children with developmental disabilities. *Developmental Medicine & Child Neurology*, 57(2), 120-136. doi:10.1111/dmcn.12623
- Spruyt, K., & Gozal, D. (2011). Sleep disturbances in children with attention-deficit/hyperactivity disorder. *Expert Review of Neurotherapeutics*, 11(4), 565-577. doi:10.1586/ern.11.7
- Srivastava, C. (2013). Autism spectrum disorders: Increasing prevalence and unmet need. *Journal of Indian Association for Child and Adolescent Mental Health*, 9(3), 52-61.

- Stanley, G. C., & Konstantareas, M. M. (2007). Symbolic play in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37(7), 1215-1223. doi:10.1007/s10803-006-0263-2
- Stoléru, S., Nottelmann, E. D., Belmont, B., & Ronsaville, D. (1997). Sleep problems in children of affectively ill mothers. *Journal of Child Psychology and Psychiatry*, 38(7), 831-841. doi:10.1111/j.1469-7610.1997.tb01601.x
- Stores, G. (1996). Practitioner review: Assessment and treatment of sleep disorders in children and adolescents. *Journal of Child Psychology and Psychiatry*, 37(8), 907-925. doi:10.1111/j.1469-7610.1996.tb01489.x
- Stores, G., & Stores, R. (2013). Sleep disorders and their clinical significance in children with down syndrome. *Developmental Medicine & Child Neurology*, 55(2), 126-130.
- Stores, R., & Stores, G. (2004). Evaluation of brief group-administered instruction for parents to prevent or minimize sleep problems in young children with down syndrome. *Journal of Applied Research in Intellectual Disabilities*, 17(2), 61-70. doi:10.1111/j.1360-2322.2004.00174.x
- Strang, J. F., Kenworthy, L., Daniolos, P., Case, L., Wills, M. C., Martin, A., & Wallace, G. L. (2012). Depression and anxiety symptoms in children and adolescents with autism spectrum disorders without intellectual disability. *Research in Autism Spectrum Disorders*, 6(1), 406-412. doi:10.1016/j.rasd.2011.06.015
- Sukhodolsky, D. G., Scahill, L., Gadow, K. D., Arnold, L. E., Aman, M. G., McDougle, C. J., . . . Vitiello, B. (2008). Parent-rated anxiety symptoms in children with pervasive developmental disorders: Frequency and association with core autism symptoms and cognitive functioning. *Journal of Abnormal Child Psychology*, 36(1), 117-128. doi:10.1007/s10802-007-9165-9
- Sullivan, K. M. (2008). The interaction of agricultural pesticides and marginal iodine nutrition status as a cause of autism spectrum disorders. *Environmental Health Perspectives*, 116(4), A155-A155. doi:10.1289/ehp.11010
- Symons, F. J., Davis, M. L., & Thompson, T. (2000). Self-injurious behavior and sleep disturbance in adults with developmental disabilities. *Research in Developmental Disabilities*, 21(2), 115-123. doi:https://doi.org/10.1016/S0891-4222(00)00028-7
- Talbott, E. O., Arena, V. C., Rager, J. R., Clougherty, J. E., Michanowicz, D. R., Sharma, R. K., & Stacy, S. L. (2015). Fine particulate matter and the risk of autism spectrum disorder. *Environmental Research*, 140, 414-420. doi:https://doi.org/10.1016/j.envres.2015.04.021

- Tani, P., Lindberg, N., Nieminen-von Wendt, T., von Wendt, L., Alanko, L., Appelberg, B., & Porkka-Heiskanen, T. (2003). Insomnia is a frequent finding in adults with asperger syndrome. *BMC psychiatry*, 3(1), 12-12. doi:10.1186/1471-244X-3-12
- Tani, P., Lindberg, N., Nieminen-von Wendt, T., von Wendt, L., Virkkala, J., Appelberg, B., & Porkka-Heiskanen, T. (2004). Sleep in young adults with asperger syndrome. *Neuropsychobiology*, 50(2), 147-152. doi:10.1159/000079106
- Taylor, M. A., Schreck, K. A., & Mulick, J. A. (2012). Sleep disruption as a correlate to cognitive and adaptive behavior problems in autism spectrum disorders. *Research in Developmental Disabilities*, 33(5), 1408-1417. doi:10.1016/j.ridd.2012.03.013
- Thorpy, M. J. (2012). Classification of sleep disorders. *Neurotherapeutics*, 9(4), 687-701. doi:10.1007/s13311-012-0145-6
- Thunstrom, M., & Ferber, R. (1999). Severe sleep problems among infants: Family and infant characteristics. *Ambulatory Child Health*, 5(1), 27-41.
- Tilford, J. M., Payakachat, N., Kuhlthau, K. A., Pyne, J. M., Kovacs, E., Bellando, J., . . . Frye, R. E. (2015). Treatment for sleep problems in children with autism and caregiver spillover effects. *Journal of Autism and Developmental Disorders*, 45(11), 3613-3623. doi:10.1007/s10803-015-2507-5
- Tordjman, S., Najjar, I., Bellissant, E., Anderson, G., Barbueroth, M., Cohen, D., . . . Vernay-Leconte, J. (2013). Advances in the research of melatonin in autism spectrum disorders: Literature review and new perspectives. *International Journal of Molecular Sciences*, 14(10), 20508-20542. doi:10.3390/ijms141020508
- Tordjman, S., Somogyi, E., Coulon, N., Kermarrec, S., Cohen, D., Bronsard, G., . . . Xavier, J. (2014). Gene \times environment interactions in autism spectrum disorders: Role of epigenetic mechanisms. *Frontiers in Psychiatry*, 5(53). doi:10.3389/fpsy.2014.00053
- Tudor, M. E., Hoffman, C. D., & Sweeney, D. P. (2012). Children with autism: Sleep problems and symptom severity. *Focus on Autism and Other Developmental Disabilities*, 27(4), 254-262. doi:10.1177/1088357612457989
- Turnbull, K., Reid, G. J., & Morton, J. B. (2013). Behavioral sleep problems and their potential impact on developing executive function in children. *Sleep*, 36(7), 1077-1084. doi:10.5665/sleep.2814
- Turner, K. S., & Johnson, C. R. (2013). Behavioral interventions to address sleep disturbances in children with autism spectrum disorders: A review. *Topics in Early Childhood Special Education*, 33(3), 144-152. doi:10.1177/0271121412446204

- Vargas, D. L., Nascimbene, C., Krishnan, C., Zimmerman, A. W., & Pardo, C. A. (2005). Neuroglial activation and neuroinflammation in the brain of patients with autism. *Annals of Neurology*, 57(1), 67-81. doi:10.1002/ana.20315
- Varni, J., Seid, M., & Kurtin, P. (1998). Pediatric quality of life inventory version 4.0. *Lyon, France: Mapi Research Institute.*
- Veatch, O. J., Sutcliffe, J. S., Warren, Z. E., Keenan, B. T., Potter, M. H., & Malow, B. A. (2017). Shorter sleep duration is associated with social impairment and comorbidities in asd. *Autism research*, 10(7), 1221-1238. doi:10.1002/aur.1765
- Volk, H. E., Hertz-Picciotto, I., Delwiche, L., Lurmann, F., & McConnell, R. (2011). Residential proximity to freeways and autism in the charge study. *Environmental Health Perspectives*, 119(6), 873.
- Volk, H. E., Kerin, T., Lurmann, F., Hertz-Picciotto, I., McConnell, R., & Campbell, D. B. (2014). Autism spectrum disorder: Interaction of air pollution with the met receptor tyrosine kinase gene. *Epidemiology*, 25(1), 44-47. doi:10.1097/ede.0000000000000030
- Volkmar, F. R., Szatmari, P., & Sparrow, S. S. (1993). Sex differences in pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 23(4), 579-591. doi:10.1007/BF01046103
- Vriend, J., & Corkum, P. (2011). Clinical management of behavioral insomnia of childhood. *Psychology research and behavior management*, 4, 69-79. doi:10.2147/PRBM.S14057
- Vriend, J. L., Corkum, P. V., Moon, E. C., & Smith, I. M. (2011). Behavioral interventions for sleep problems in children with autism spectrum disorders: Current findings and future directions. *Journal of Pediatric Psychology*, 36(9), 1017-1029. doi:10.1093/jpepsy/jsr044
- Wachtel, K., & Carter, A. S. (2008). Reaction to diagnosis and parenting styles among mothers of young children with asds. *Autism*, 12(5), 575-594. doi:10.1177/1362361308094505
- Wake, M., Morton-Allen, E., Poulakis, Z., Hiscock, H., Gallagher, S., & Oberklaid, F. (2006). Prevalence, stability, and outcomes of cry-fuss and sleep problems in the first 2 years of life: Prospective community-based study. *Pediatrics*, 117(3), 836-842.

- Weisbrot, D. M., Gadow, K. D., DeVincent, C. J., & Pomeroy, J. (2005). The presentation of anxiety in children with pervasive developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 15(3), 477-496. doi:10.1089/cap.2005.15.477
- Weiskop, S., Matthews, J., & Richdale, A. (2001). Treatment of sleep problems in a 5-year-old boy with autism using behavioural principles. *Autism*, 5(2), 209-221. doi:10.1177/1362361301005002009
- Weiskop, S., Richdale, A. L., & Mathews, J. (2005). Behavioural treatment to reduce sleep problems with autism and fragile x syndrome. *Developmental Medicine and Child Neurology*, 47(2), 94-104. doi:10.1017/s0012162205000186
- Werling, D. M., & Geschwind, D. H. (2013). Sex differences in autism spectrum disorders. *Current opinion in neurology*, 26(2), 146-153. doi:10.1097/WCO.0b013e32835ee548
- Werner, E., Dawson, G., Munson, J., & Osterling, J. (2005). Variation in early developmental course in autism and its relation with behavioral outcome at 3–4 years of age. *Journal of Autism and Developmental Disorders*, 35(3), 337-350. doi:10.1007/s10803-005-3301-6
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clin Psychol Rev*, 29(3), 216-229. doi:10.1016/j.cpr.2009.01.003
- Wiggs, L. (2001). Sleep problems in children with developmental disorders. *Journal of the Royal Society of Medicine*, 94(4), 177-179.
- Wiggs, L. (2007). Are children getting enough sleep? Implications for parents. *Sociological Research Online*, 12(5), 1-16. doi:10.5153/sro.1557
- Wiggs, L., & Stores, G. (1996). Severe sleep disturbance and daytime challenging behaviour in children with severe learning disabilities. *Journal of Intellectual Disability Research*, 40(6), 518-528. doi:10.1046/j.1365-2788.1996.799799.x
- Wiggs, L., & Stores, G. (2001). Behavioural treatment for sleep problems in children with severe intellectual disabilities and daytime challenging behaviour: Effect on mothers and fathers. *British Journal of Health Psychology*, 6(3), 257-269. doi:10.1348/135910701169197
- Wiggs, L., & Stores, G. (2004). Sleep patterns and sleep disorders in children with autistic spectrum disorders: Insights using parent report and actigraphy. *Developmental Medicine and Child Neurology*, 46(6), 372-380. doi:10.1017/s0012162204000611

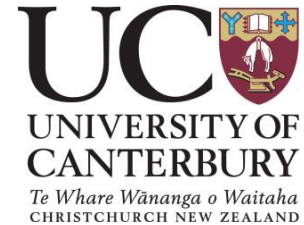
- Wijngaarden-Cremers, P. J. v., Eeten, E. v., Groen, W. B., Deurzen, P. A. M. v., Oosterling, I. J., & Gaag, R. J. v. d. (2014). Gender and age differences in the core triad of impairments in autism spectrum disorders: A systematic review and meta-analysis. *Journal of Autism and Developmental Disorders*, 44(3), 627-635. doi:10.1007/s10803-013-1913-9
- Williams, J. H. G., Whiten, A., Suddendorf, T., & Perrett, D. I. (2001). Imitation, mirror neurons and autism. *Neuroscience & Biobehavioral Reviews*, 25(4), 287-295.
- Williams, K., Woolfenden, S., Roberts, J., Rodger, S., Bartak, L., & Prior, M. (2014). Autism in context 1: Classification, counting and causes. *Journal of Paediatrics and Child Health*, 50(5), 335-340. doi:10.1111/jpc.12451
- Williams, K. C., Fuchs, G. J., Furuta, G. T., Marcon, M. A., & Coury, D. L. (2010). Clinical features associated with gi symptoms in autism spectrum disorders (ASD). *Gastroenterology*, 138(5), S-74.
- Williams, P. G., Sears, L. L., & Allard, A. (2004). Sleep problems in children with autism. *Journal of Sleep Research*, 13(3), 265-268. doi:10.1111/j.1365-2869.2004.00405.x
- Wilshire, C. (2017). The problem of bias in behavioural intervention studies: Lessons from the pace trial. *Journal of Health Psychology*, 22(9), 1128-1133. doi:10.1177/1359105317700885
- Wolf, M., Risley, T., Johnston, M., Harris, F., & Allen, E. (1967). Application of operant conditioning procedures to the behavior problems of an autistic child: A follow-up and extension. *Behaviour Research and Therapy*, 5(2), 103-111. doi:10.1016/0005-7967(67)90004-6
- Wolf, M., Risley, T., & Mees, H. (1963). Application of operant conditioning procedures to the behaviour problems of an autistic child. *Behaviour Research and Therapy*, 1(2-4), 305-312.
- Wolfson, A., Lacks, P., & Futterman, A. (1992). Effects of parent training on infant sleeping patterns, parents' stress, and perceived parental competence. *Journal of Consulting and Clinical Psychology*, 60(1), 41.
- Wong, K., Leonard, H., Jacoby, P., Ellaway, C., & Downs, J. (2015). The trajectories of sleep disturbances in rett syndrome. *Journal of Sleep Research*, 24(2), 223-233. doi:10.1111/jsr.12240
- Xu, G., Jing, J., Bowers, K., Liu, B., & Bao, W. (2014). Maternal diabetes and the risk of autism spectrum disorders in the offspring: A systematic review and meta-analysis.

Journal of Autism and Developmental Disorders, 44(4), 766-775.
doi:10.1007/s10803-013-1928-2

- Yang, X. L., Liang, S., Zou, M. Y., Sun, C. H., Han, P. P., Jiang, X. T., . . . Wu, L. J. (2018). Are gastrointestinal and sleep problems associated with behavioral symptoms of autism spectrum disorder? *Psychiatry Res*, 259, 229-235.
doi:10.1016/j.psychres.2017.10.040
- Zerbo, O., Iosif, A.-M., Walker, C., Ozonoff, S., Hansen, R. L., & Hertz-Picciotto, I. (2013). Is maternal influenza or fever during pregnancy associated with autism or developmental delays? Results from the charge (childhood autism risks from genetics and environment) study. *Journal of Autism and Developmental Disorders*, 43(1), 25-33. doi:10.1007/s10803-012-1540-x
- Zerbo, O., Qian, Y., Yoshida, C., Grether, J. K., Van de Water, J., & Croen, L. A. (2015). Maternal infection during pregnancy and autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 45(12), 4015-4025.
- Zhu, L., Wang, X., Li, X.-L., Towers, A., Cao, X., Wang, P., . . . Jiang, Y.-H. (2014). Epigenetic dysregulation of shank3 in brain tissues from individuals with autism spectrum disorders. *Human Molecular Genetics*, 23(6), 1563-1578.
doi:10.1093/hmg/ddt547
- Zuckerman, K. E., Hill, A. P., Guion, K., Voltolina, L., & Fombonne, E. (2014). Overweight and obesity: Prevalence and correlates in a large clinical sample of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(7), 1708-1719. doi:10.1007/s10803-014-2050-9
- Zwaigenbaum, L. (2001). Autistic spectrum disorders in preschool children. *Canadian Family Physician*, 47(10), 2037-2042.
- Zwaigenbaum, L., Bryson, S., Lord, C., Rogers, S., Carter, A., Carver, L., . . . Yirmiya, N. (2009). Clinical assessment and management of toddlers with suspected autism spectrum disorder: Insights from studies of high-risk infants. *Pediatrics*, 123(5), 1383-1391. doi:10.1542/peds.2008-1606
- Zwaigenbaum, L., Bryson, S. E., Szatmari, P., Brian, J., Smith, I. M., Roberts, W., . . . Roncadin, C. (2012). Sex differences in children with autism spectrum disorder identified within a high-risk infant cohort. *Journal of Autism and Developmental Disorders*, 42(12), 2585-2596. doi:10.1007/s10803-012-1515-y

Appendices

Appendix A: Child Information Sheet



An investigation into the effectiveness of treatments for sleep disturbance in children with autism

Children's Information Sheet



Hello. My name is XXX and I am a teacher/student/psychologist at the University of Canterbury. I am doing a project about how to help children to sleep better and I would like for you to help me with this.

I am going to be talking to you and your parent/s about ways to help you to sleep better. This means that I might be Skyping you, coming to your house, or your parent/s will be coming to see me at the University.

There will be a video camera in your bedroom sometimes. This will help me to understand what you do when you are awake and asleep. Only your parents and other people working on this project will be able to see this video. We may ask you to wear an actigraph. An actigraph is worn on your wrist like a watch and it tells us when you are asleep and when you are awake. XX, a research

assistant/Masters/PhD student who also works as a part of our sleep team, may look at some of the information that we collect, such as video recordings and actigraph data.

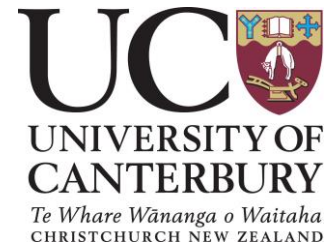
If you do not want to be a part of this project, you can tell me or your parents and you won't need to be a part of it anymore.

If you have any questions you can ask me or your parents whenever you like.

Now we need to decide if you would like to do this. If you do want to be a part of my project then you can say "yes". If you do not want to be a part of this project then you can say "no" and no one will mind.

If you say yes, you or one of your parents can sign the form for you.

Appendix B: Child Consent Form



“An investigation into the efficacy of treatments for sleep disturbance in children with autism”

Children’s Consent Form

My name is _____.

- ☐ XXX has told me about the work that she is going to be doing with me and my parent/s.
- ☐ XXX told me that she is going to be working with me and my parent/s to help me to learn to sleep better.
- ☐ While XXX does this she will be asking my parents about my sleep each night and there will be a video camera in my room on some nights that is recording my sleep.
- ☐ I know that if at any time I want to stop being a part of this project then XXX will stop recording data and this will be destroyed.
- ☐ If I want XX to stop video recording my sleep then the camera will be taken out of my room and that will be fine. If I want any video footage to be deleted, I can tell XXX or my parents.
- ☐ I was told that my parents/caregiver may sign this form for me and I think that is OK.
- ☐ I would like a summary of the results of this project.

Child’s name: _____

Date: _____

Signature: _____

If this form is signed on behalf of your child please acknowledge, by signing this form, that your child was verbally informed of the investigation and what it will involve and that they were unable to provide verbal or written consent that they would like to be a part of this research.

Parent/caregiver: _____

Date: _____

Signature: _____

Please return this form to XXX.

Appendix C: Parent Information Sheet

An investigation into the effectiveness of treatments for sleep disturbance in children with autism or features of autism

Information for Parents/Caregivers

This research has been assessed and approved by the University of Canterbury Human Ethics Committee (HEC 2018/47).

Dear Parent/ Caregiver,

We are a group of researchers at the University of Canterbury. Dr Laurie McLay is a Senior Lecturer in the School of Health Sciences at the University of Canterbury. Laurie has many years experience in working with children and young people with developmental disabilities and their families. Associate Professor Karyn France has lectured here for many years, has conducted research into the treatment of paediatric sleep disturbance and is a registered clinical psychologist with considerable clinical experience in this area. Professor Neville Blampied has a similar history of teaching and research. A number of Masters and PhD students and Child and Family Intern psychologists or registered psychologist also work on this project.

We would like you and your child with autism to consider participating in this research study. The primary purpose of this study is to investigate the effectiveness of treatments for sleep disturbance in children with autism. Treatment can include a range of strategies, including both non-traditional approaches (such as white noise) and behavioural interventions. These approaches have been designed to minimise stress as much as possible for the parents and children using them. We are also interested in parents' and children's experiences in using the treatments and any changes to their lives, or their child's lives, which result.

As a part of this study we would also like to investigate the experiences of parents in implementing treatments for sleep disturbance, those treatments that they consider to be most acceptable, and the impact of successful treatment of sleep problems on parent and child wellbeing and quality of life. In order to do this we will ask you to complete some questionnaires about you and your child's well-being and behaviour at the commencement and conclusion of treatment. We will also ask your perspective on the treatment that was

provided. We will do this either during visits to your home, Skype interviews, or in a clinic at the University of Canterbury.

If you agree to allow your child to be a part of this study, we will meet with you, or Skype you, to discuss your child's sleep behaviour and find out more about him/her and your family. This initial meeting will last for approximately 1-1 ½ hours. We will then ask you to complete sleep diaries in which you will record further information about your child's sleep patterns. Sleep diaries will be recorded each day throughout all phases of the study as this will allow us to monitor the effectiveness of the treatment approach. The sleep diaries will take you up to five minutes to complete each night. You will also be asked to complete commonly used questionnaires in order to obtain information about your child's sleep behaviour and the effects of treatment. It will take approximately 15 minutes to complete each questionnaire. When we have established an understanding of your child's sleep behaviour, we will work with you to develop sleep-related goals for your child. This will involve a second treatment planning session which will last 1-1 ½ hours.

To help us gather further information about your child's sleep patterns we will bring or send a video camera to your home for some nights over the course of the programme, which is capable of recording all night sleep. In addition we may ask you, if possible, to use an actigraph with your child. This watch-like device records the movements associated with sleep and can be worn on the wrist or ankle, or secured into a pocket on your child's pyjamas. This may offer an alternative to video cameras when appropriate. These methods will allow us to measure sleep behaviour at times when an adult is not present. We will demonstrate and explain how to use each of these methods for gathering information.

When information about your child's sleep behaviour has been gathered, treatment will commence. You will be offered a choice of treatment options, which you will then implement with the support of the research team. If you are dissatisfied with the treatment approach or the degree of progress that is being made then you will be offered a choice of another treatment option. We will provide you with all of the necessary information about each treatment approach and we will maintain regular contact with you during treatment. It is anticipated that your involvement in the study will occur over the course of a few months, but will depend on the rate of your child's progress as well as your satisfaction with the progress.

For the purpose of this project, myself (insert name), a psychologist/intern psychologist/Masters/PhD student will be working closely with you to conduct the necessary assessments and formulate interventions. XX, a research assistant/Masters/PhD student who also works as a part of our sleep team, may look at some of the information that we collect, such as video recordings and actigraph data.

Your child will be assigned a code name to ensure anonymity and anything that you or your child says or does will be kept confidential. The results of the study may be submitted for publication to national or international journals and may also be presented at conferences. No identification of the child or family will be possible from any report, publication or presentation.

If you want to withdraw from the project before completion, you can do this at any time without penalty or repercussions.

Should you require any additional information about the study or if you would like to access the study findings you are able to do so at any stage. The data which is produced from the research will be kept in a locked cabinet at the University of Canterbury for a minimum of ten years.

If you agree for your child to take part in the research, please sign the consent form that is attached.

If you have any complaints you may contact the Chair of the University of Canterbury Ethics Committee. The contact details are given below.

If you have any questions about this project please feel free to contact Dr Laurie McLay: Phone (03) 369-3522 or, email: laurie.mclay@canterbury.ac.nz

Appendix D: Parent Consent Form



An investigation into the effectiveness of treatments for sleep disturbance in children with autism

CONSENT FORM FOR PARENTS/ CAREGIVERS

This research has been assessed and approved by the University of Canterbury, Human Ethics Committee (HEC 2018/47).

- ☐ I wish to participate in the project, “An investigation into the efficacy of treatments for sleep disturbance in children with autism”
- ☐ I have read and been given a full explanation of this project and have had the opportunity to ask questions.
- ☐ I understand what will be required of myself and my child/the child in my care during this project.
- ☐ I understand that the investigators do not foresee any potential risks to me or my child as a result of participating in this study. However, if the intervention results in an increase in family stress, the staff working with us will provide support.
- ☐ I understand that all information about my family will be treated as confidential unless there is concern about anyone’s safety. In this case my clinician will need to speak to someone else to ensure the safety risk is removed. No findings that could identify me or my child will be published
- ☐ I understand that the findings of this study may be published in a research journal or at a conference and that the anonymity of my child and I will be maintained
- ☐ I understand that participation in this project is voluntary and that I can withdraw my child or he/she can withdraw from the project at any time without repercussions. I can also withdraw any data that has been collected at any time prior to the publication of that data

- ☐ I understand that all research data that is collected will be securely stored at the University of Canterbury for a minimum of ten years
- ☐ I understand that I am able to request a copy of the results of this research, should I wish to do so, and that these results will be provided for me
- ☐ I allow video-taping of my child's sleep behaviour to be completed by the researcher and understand that this videotape will be used for data gathering purposes only. I also understand that I have the right to request that video footage is destroyed at any stage.
- ☐ I consent to others, listed below, being involved in the implementation of the intervention

Name: _____

Date: _____

Signature: _____

Others I consent to implementing intervention:

Name: _____

Name: _____

Name: _____

- ☐ I would like a summary of the results of this project.

Please return this form to XXX

Appendix E: Sleep Diary Template

	Date:	Monday:	Tuesday:	Wednesday:	Thursday:	Friday:	Saturday:	Sunday:
Daytime sleep	Setting (where fell asleep)							
	Time asleep							
	Time awake							
Night-time sleep	Setting (where fell asleep)							
	Time put to bed							
	Frequency of Curtain calls*							
	Curtain calls after put to bed (Describe each)							
	Your responses to each curtain call (Describe each)							
	Best estimate of time asleep							

[illegible]

Time awake in the morning								
---------------------------	--	--	--	--	--	--	--	--

Notes: *Curtain calls: Any behaviour such as leaving the bed (or bedroom) or calling parents into the room, between the time of being put to bed and falling asleep